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RESPIRATORY CARE

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38TH YEAR—ESTABLISHED 1956

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- Pulmonary Compliance via Volume-Controlled Infant Resuscitator: A Bench Study
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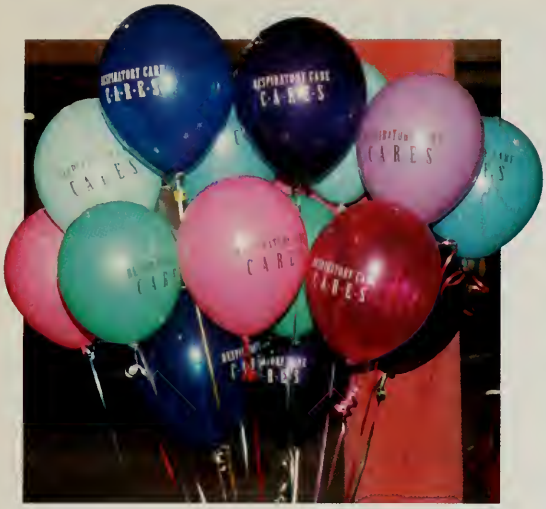

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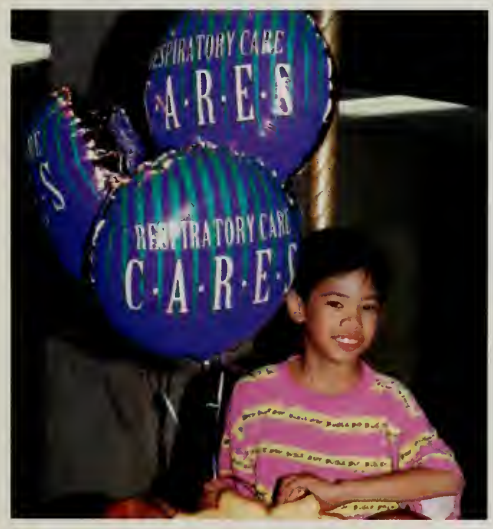

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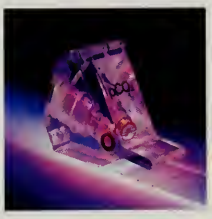

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What's the Denominator? (editorial)—CG Victora. The Lancet 1993;342:97.

Pandemonium in the Modern Hospital (editorial)—A Jacobson. N Engl J Med 1993;329:211.

Australian View of Paediatric Intensive Care in Britain (editorial)—F Shann. The Lancet 1993;342:68.

A Computer-Controlled Ventilator Weaning System: A Clinical Trial—JH Strickland Jr, JH Hasson. Chest 1993;103:1220.

The purpose of this study was to determine if a computer-directed weaning system could wean patients with complex medical problems from mechanical ventilation and appropriately respond to signs of respiratory failure more effectively than traditional physician-directed weaning methods. In a prospective, randomized, controlled study, we tested the system in 15 patients who had required prolonged mechanical ventilation and met predetermined weaning tests. Patients were randomly assigned to one of two groups: (1) automatic, computer-directed weaning or (2) physician-controlled weaning. The computer-directed system decreased or increased the synchronized intermittent mandatory ventilation rate and pressure support (PS) based on predetermined limits of patient respiratory rate (RR) and tidal volume (TV). Pulse oximeter oxygen saturation was monitored, and alarms were triggered by an oxygen saturation of < 90 percent. In the physician control group, weaning progressed with SIMV rate and PS reduction, as judged appropriate by the physician. Nine patients were assigned to the computer group; six patients were assigned to the control group. The average patient age was 64.8 ± 14.9

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years for the computer group, 65.2 ± 22.7 years for the control group. The average time on mechanical ventilation prior to weaning was 13.4 ± 7.8 days for the computer group and 14.5 ± 11.1 days for the control group. Seven of the nine computer group patients weaned within 48 h of the study, with an average time to wean of 18.7 ± 5.9 h. All seven were breathing spontaneously 48 h after weaning. Two of the six control group patients weaned within 48 h of the study, with an average time of 25.6 ± 5.6 h. Both patients who weaned were placed back on mechanical ventilation within 30 h. The number of arterial blood gas samples drawn during the study was 1.4 ± 0.7 for the computer group, 7.2 ± 4.3 for the control group. The number of minutes per hour outside acceptable limits of RR > 30, RR < 8, or TV< 5 mL/kg was 3.2 ± 2.8 min for the computer group and 6.6 ± 4.1 min for the control group. The study suggests that use of the computer-directed weaning system results in fewer arterial blood gas samples, shorter weaning times, and less time spent outside acceptable RR and TV parameters.

Effects of Short-Term Oxygenation Changes on Acute Lung Injury Patients Undergoing Pressure Support Ventilation—A Pesenti, N Rossi, A Calori, G Foti, GP Rossi. Chest 1993;103:1185.

We investigated the effects of short-term oxygenation changes upon the neuromuscular respiratory drive (airway occlusion pressure [$P_{0.1}$]), minute ventilation ($\dot{V}_E$), and respiratory rate (RR) in 12 acute lung injury patients undergoing pressure support ventilation. We ventilated the patients first at a high level (H1) of oxygenation, then at intermediate (I), at low, (L) and again at the high (H2) level. The H1 and H2 periods showed no differences. In the H1, I, and L periods, P_{aO_2} was 158 ± 68, 75 ± 12, and 55 ± 6 mm Hg, respectively. Decreasing oxygenation caused very significant increases in $\dot{V}_E$, RR, and $P_{0.1}$. Differences in RR, $\dot{V}_E$, and rapid shallow breathing index were significant at step H1 versus I. Changes in $P_{0.1}$ appeared to be higher when the H1 value was higher than normal. An arterial oxygenation target higher than the generally accepted 60 mm Hg level may decrease both RR and $\dot{V}_E$.

Chronic Obstructive Pulmonary Disease Mortality: A Role for Altitude—TR Coté, DF Stroup, DM Dwyer, JM Horan, DE Peterson. Chest 1993; 103:1194.

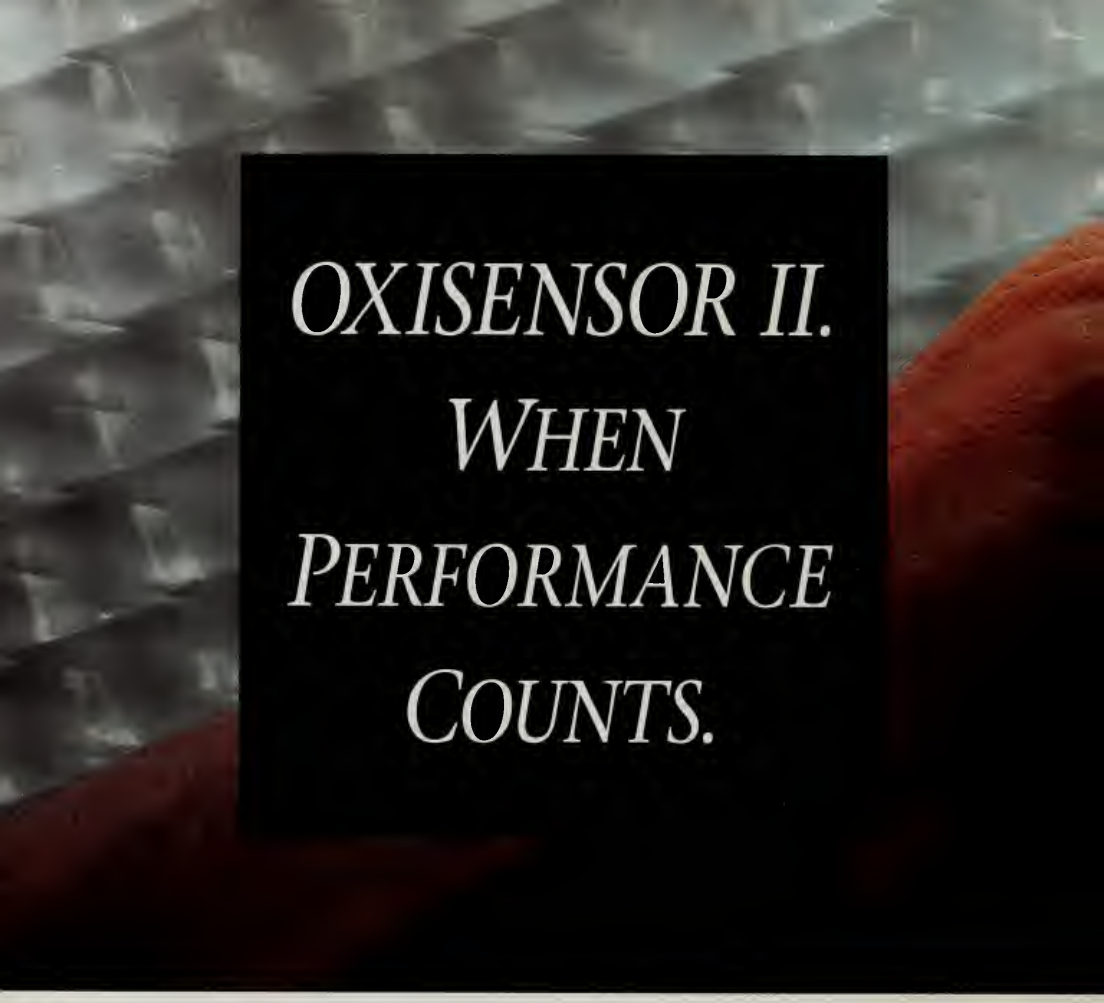
A map of U.S. COPD mortality rates by state suggested that the relative hypoxia of increased altitude may be independently associated with COPD mortality. This was investigated using linear regression analysis of 1986 state-specific data on COPD mortality rates, history of cigarette consumption, and altitude. County seat altitudes and county populations were used to calculate the median altitude of state residents. We found independent significant associations between COPD and both smoking and altitude. State COPD mortality rose by $1/10^5$ for every 5.4 increase in mean packs consumed per capita per year or for each 95-m increase in resident altitude. There was no association between altitude and smoking. If increased altitude does contribute to COPD mortality, persons with this disease may benefit from down-migration.

Prediction of Minimal Pressure Support during weaning from Mechanical Ventilation—SD Nathan, AM Ishaaya, SK Koerner, MJ Belman. Chest 1993;103:1215.

Our goal was to evaluate the accuracy of a prediction equation that estimates the minimal level of pressure support (PS) required to overcome the resistance of the ventilator circuit and the endotracheal tube during mechanical ventilation. We calculated the minimal PS (PSmin) by means of the relationship between airway resistance and peak inspiratory flow rate during mechanical ventilation. Measurements of breathing pattern, flow rates, work of breathing (WOB), pressure time product (PTP), and PEEPi were made during several modes of ventilation (PSmin, PSmin + 25 percent, PSmin-25 percent, flow by, CPAP 0 cm H_2O) and while breathing through an endotracheal tube (ETT) and spontaneous breathing (EXT). The WOB was significantly higher during EXT than PSmin, PSmin-25 percent, and ETT (1.04 vs 0.45, 0.54, and 0.74 J/L, respectively, $p < 0.05$). An unexpected finding was a higher WOB and PTP during EXT as compared with ETT in six of seven of our patients (1.04 vs 0.74 J/L). Examination of breathing pattern and flow volume loops in these two breathing modes raises the possibility that the post-EXT pathology increases in WOB are related to upper airway abnormality. Because of this, our predicted PSmin underestimated the WOB required for spontaneous breathing immediately post EXT.

Controlled Trial of a Continuous Irrigation Suction Catheter vs Conventional Intermittent Suction Catheter in Clearing Bronchial Secretions from Ventilated Patients—JO Isea, D Poyant, C O'Donnell, LJ Faling, J Karlinsky, BR Celli. Chest 1993;103:1227.

Continuous irrigation-suction catheter (Irri-cath) is a double-lumen device that allows for simultaneous saline solution infusion and aspiration. This system may theoretically be more effective than conventional dry intermittent suction due to its vortex



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principle. To test this hypothesis, we performed 200 suction maneuvers in 20 ventilated patients. Identically shaped catheters were used in a randomized sequence. For the same individual, we used equal instilled saline solution volume (40 mL), vacuum pressure (–180 cm H_2O), and ventilatory parameters. Effectiveness of suction was determined by measuring the total aspirated volume, the dry lyophilized weight of secretion, the corrected dry weight (dry weight – weight of instilled salt), and protein concentration. No difference in heart rate, respiratory frequency, O_2 saturation, systemic blood pressure, peak inspiratory pressure, or patient discomfort was found when the two modalities were compared; however, the total volume of secretions collected, the dry weight, the corrected dry weight, and the protein concentration were significantly higher with continuous irrigation suction catheter when compared with the conventional method ($p < 0.05$). The suction time was shorter with the Irri-cath ($p < 0.05$). We conclude that the Irri-Cath is more effective than conventional intermittent suction catheter in clearing bronchial secretions in patients on mechanical ventilation.

Lung Lavage with Oxygenated Perfluorochemical Liquid in Acute Lung Injury—PS Richman, MR Wolfson, TH Shaffer. Crit Care Med 1993;21: 768.

Objective: To investigate the effects of lung lavage with oxygenated liquid perfluorochemical on gas exchange, lung mechanics, and cardiac function in animals with acute lung injury. Design: Prospective, randomized, controlled trial. Setting: Animal laboratory. Subjects: Eight adult cats (2 to 4 kg, random sex). Interventions: Two insults were combined to cause lung injury: oleic acid infusion and saline whole-lung wash. Animals were assigned to either the control or treatment group which consisted of a perfluorochemical liquid (Rimar 101) lavage. Perfluorochemical liquid lavage was performed three times at hourly intervals after lung injury. Three other cats with identical injury but no perfluorochemical liquid lavage served as control animals. All cats were ventilated with an F_{IO_2} of 0.95 and positive end-expiratory pressure of 2 cm H_2O continuously. Measurements and Main Results: Arterial blood gas tensions and pH, dynamic pulmonary compliance were measured at 15-min intervals. Cardiac index was assessed hourly, and lung fluid was collected after each of the three perfluorochemical liquid lavages. Arterial oxygen tension and pulmonary compliance deteriorated abruptly after lung injury in all cats, and improved significantly ($p < 0.001$, two-way analysis of variance) 15 mins after perfluorochemical liquid lavage. These parameters gradually returned to their baseline over 60 mins. Arterial blood pressure and cardiac index decreased after injury in all cats, and were not significantly changed after perfluorochemical liquid lavage. Hemorrhagic fluid was recovered from distal airways by perfluorochemical liquid lavage, despite prior suctioning of the airway. Conclusions: Perfluorochemical liquid lavage removes pulmonary edema fluid and improves gas exchange and the mechanical properties of the lung, after acute severe lung injury.

Preventing Bronchoconstriction in Exercise-Induced Asthma with Inhaled Heparin—T Ahmed, J Garrigo, I Danta. N Engl J Med 1993; 329:90.

Background: We have previously reported that inhaled heparin prevents allergic bronchoconstriction in sheep and inhibits the anti-IgE-mediated release of histamine from mast cells in vitro. Since the release of such mediators has been implicated in exercise-induced asthma, we investigated whether inhaled heparin could also attenuate the bronchoconstrictor response in this disease. Methods: On five days we studied 12 subjects with a history of exercise-induced asthma. On day 1 they underwent a standardized exercise challenge on a treadmill to document the presence of exercise-induced asthma. Minute ventilation was estimated with a calibrated respiratory inductive plethysmograph. The workload was increased until the heart rate reached 85 percent of the predicted maximal value, and was sustained for 10 minutes. The magnitude of bronchoconstriction was assessed by measuring specific airway conductance before and after the exercise. On day 2 the partial-thromboplastin time was measured in plasma obtained before and after the subjects inhaled a nebulized solution of heparin (1000 U per kilogram of body weight). On days 3 through 5 the subjects were pretreated with 4 mL of inhaled heparin (1000 U per kilogram), cromolyn sodium (20 mg), or placebo according to a single-blind, randomized, crossover design and underwent exercise challenge 45 minutes later. To exclude the possibility that heparin had any direct effect on airway smooth muscle, bronchial provocation with histamine was induced in five subjects on two further days after pretreatment with either heparin or placebo. Results: Inhaled heparin and cromolyn sodium had no effect on specific airway conductance at base line, but did attenuate the exercise-induced decreases in this variable: the mean (±SE) maximal decrease five minutes after exercise was 9±5 percent after pretreatment with heparin, as compared with 22±5 percent after pretreatment with cromolyn and 35±2 percent after pretreatment with placebo. Heparin did not change the partial-thromboplastin time and did not modify the bronchoconstrictor re-

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[1]M.K. Reeves-Hoche', et al, CPAP vs. BiPAP: Patient Compliance in the Treatment of Obstructive Sleep Apnea, Six Month Data in a Two-Year Study. Presented at ALA/ATS 1993. Study funded by Respironics. [2]$p = .09$ [3]$p \leq .001$

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sponse to histamine. Conclusions: Inhaled heparin prevents exercise-induced asthma without influencing histamine-induced bronchoconstriction. This non-anticoagulant action of heparin is more likely to be related to a modulation of mediator release than to a direct effect on smooth muscle.

High-Frequency Jet Ventilation in Children with the Adult Respiratory Distress Syndrome Complicated by Pulmonary Barotrauma —DW Smith, LR Frankel, MT Derish, RR Moody, LE Black, BE Chipps, LH Mathers. Pediatr Pulmonol 1993;15-279.

High-frequency jet ventilation (HFJV) was used in 29 children with severe ARDS complicated by pulmonary barotrauma (PBT). Treatment with HFJV was begun when PBT was progressing over a 24-h period while receiving conventional ventilation (CV). The mean (±SD) age was 0.95 ± 1.21 years (range, 0.03-4 years). The most common diagnosis was viral pneumonia (n = 17); other diagnoses included aspiration pneumonitis (n = 4), bacterial pneumonia (n = 3), multiple trauma (n = 2), and near-drowning (n = 3). The Bunnell Life-Pulse ventilator was used at a rate of 240/min or 300/min, with inspiratory time of 0.02 sec. Twenty children survived (69%). Survivors and nonsurvivors had equal disease severity prior to HFJV as assessed by ventilator settings, alveolar-to-arterial oxygen tension gradient, oxygenation index, and blood gas values. Survivors had spent significantly less time on conventional ventilation prior to HFJV than nonsurvivors, with a mean (±SD) of 3.7 ± 2.1 days vs 9.6 ± 4.5 days, respectively ($p < 0.05$). Survivors underwent an average of 4.4 ± 3.9 days of HFJV, which supported adequate gas exchange with lower airway pressures, and produced resolution or significant improvement in airleak on chest radiograph. In conclusion, we speculate that the application of HFJV early in the course of severe hypoxemic respiratory failure complicated by airleak, allows the reduction of airway pressures, thereby minimizing pulmonary barotrauma and allowing the lung to recover from the underlying insult. Further controlled evaluation of HFJV in this high risk group of patients is warranted.

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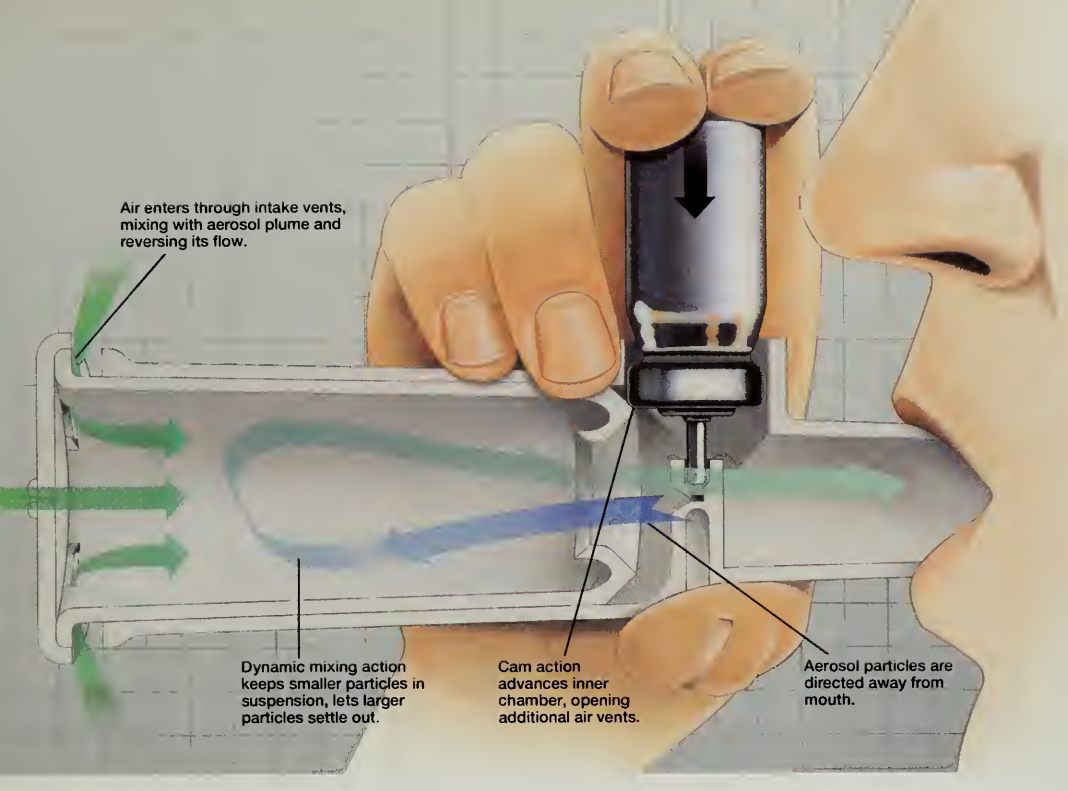
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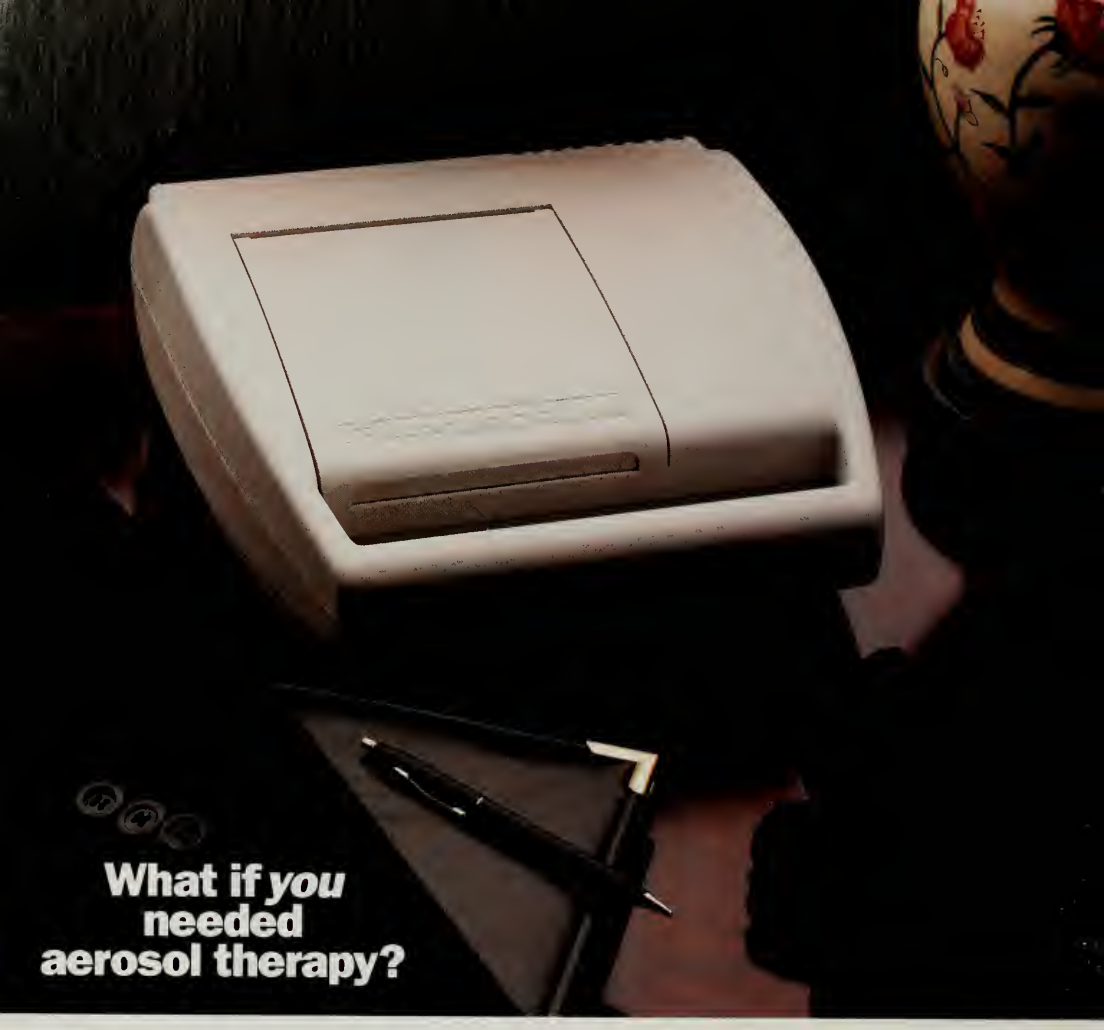

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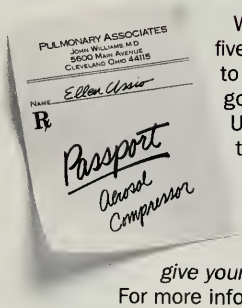

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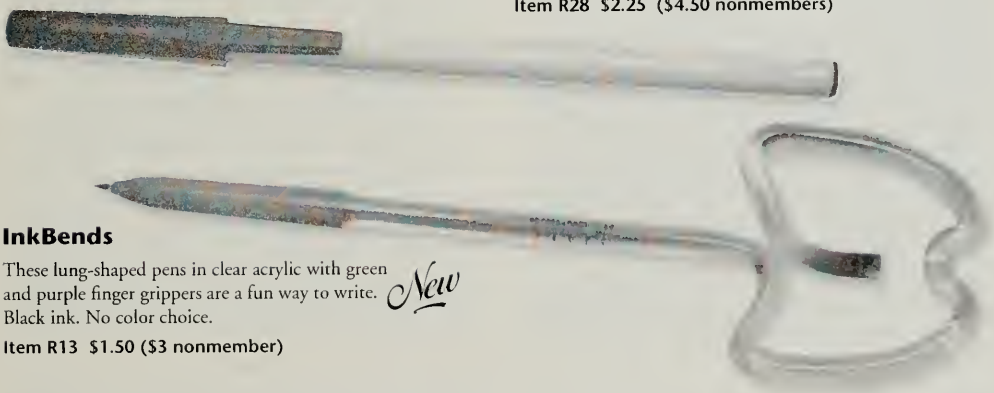

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Why We Must Help Parents Stop Smoking

A recent report in the *New England Journal of Medicine*[1] adds additional flame to the fiery issue of passive smoking's link to exacerbations of asthma. Chilmonczyk and colleagues reported that parental smoking has serious detrimental effects on children. The authors advise all of us that "the evidence that environmental tobacco smoke has a causal role in asthma-related morbidity is sufficiently strong, and the adverse pulmonary effects are sufficiently great, that systematic efforts to reduce inhalation of environmental tobacco smoke are warranted for children with asthma."[1]

Chilmonczyk and colleagues used urine cotinine levels to assess asthmatic children's exposure to environmental tobacco smoke.[1] Children with highest exposure to passive smoke measured lower on pulmonary function tests than children who were not exposed. Acute exacerbations of asthma increased with the amount of reported or measured exposure. However, reported exposure to tobacco smoke in the day-care setting did not add measurably to the children's cotinine levels achieved by exposure in the home. Would you believe—Smoking in a day-care facility? I thought it was against the law. It should be!

Earlier, Murray and Morrison[2,3] studied 415 nonsmoking asthmatic children in an allergy clinic in British Columbia. Using a symptom-severity score sheet, they found that children from households that included smokers had more problems. Lung function of these children was also worse than those from nonsmoking households. During the winter months, when windows are usually closed, lung function results were worse than in summer.[2] Air coming through open windows apparently diluted environmental tobacco smoke. Lung function and asthma severity were worst in the children exposed to their mothers' smoke. That makes sense because mothers often spend more time with their children than does anyone else.

There are numerous reports of maternal smoking and adverse consequences for the child. Maternal smoking, bottle feeding, genetic tendency, family history of respiratory allergy, and male sex predicted wheezing in children.[4] Children of mothers who smoked 10 or more cigarettes per day were 2.5 times more likely to develop asthma.[5] Women who smoked during pregnancy were likely to smoke after pregnancy.[6] A large-scale survey found that children under 5 years of age had asthma and were treated for it more often if the mother smoked.[7] Children with atopic dermatitis who had smoking mothers were 51% more likely to develop asthma if their mothers smoked.[8] Mothers are burdened with many problems; smoking mothers have the additional burden of trying to quit smoking for the health of their children. They need our advice and help. Let's give it!

In their study of the effects of passive smoking on asthmatic children, Murray and Morrison[3] found that it has a greater adverse effect on boys than on girls. In fact, the population they studied (415 pediatric asthma and wheezing patients seen consecutively at an allergy clinic) included two boys for every girl.[3]

Six years ago we read that passive smoking contributed to an increased need for emergency room visits.[9] In fact, children living with smokers visited emergency rooms 63% more often than children from nonsmoking households. The authors of that 1987 paper estimated that the average additional health-care cost for emergency care for asthma was $92 for families with one or more smokers. The impact of smoking on the family budget, as well as on national health-care costs, just keeps going up!

Reese et al[10] evaluated 468 children admitted to the general or infant ward of a children's hospital. A comprehensive respiratory questionnaire was completed, and a urine cotinine sample was obtained for each child. The authors found that children from smoking households had elevated cotinine levels. Children with bronchiolitis had higher levels of cotinine than children admitted with nonrespiratory illnesses. Another study found the same association

between parental smoking and bronchiolitis.[11] These studies make the point that we should ask all parents whether they smoke and, specifically, whether they smoke in the presence of their kids. Each child's visit or admission is an unfortunate opportunity for us to educate parents about smoking and smoking cessation.

Approximately 80% of nicotine is metabolized into cotinine. Cotinine's half-life is approximately 24 hours, so it is a useful clinical tool. Cotinine measurements are used in many smoking research studies to identify smokers, quantify nicotine intake, and verify abstinence. These tests are sensitive enough to measure environmental smoke exposure, which the commonly used exhaled carbon monoxide meter cannot do. Perhaps it is time to offer cotinine tests through the general hospital laboratory. What parent would not be moved to a good dose of guilt when told that his or her smoking measurably affects the child and that we can, in fact, measure the level of tobacco smoke exposure using cotinine tests? We could use cotinine test results as a feedback tool when working with smoking parents.

With follow-up education, we could show parents studies that relate passive smoking to increased disease in children. We could show them the associations between environmental tobacco smoke and increased hospitalization, more frequent upper respiratory tract infections, and negative impact on lung growth—just for starters.[12-20]

Can we (respiratory therapists) do this alone? No, I don't think so. We can be an active force in the hospital, pushing to develop a healthy culture that eliminates smoking. All health professionals should be role models, walking examples of good health practices. Everyone on the health team should try to help treat smokers wherever they are found. We should offer assistance to our patients (most important!), the parents of our patients, our addicted colleagues, and the general public. Developing treatment programs for smokers is the right thing to do!

Vigilante respiratory therapists will find their efforts to decrease smoking rebuffed unless the entire team agrees that it is the right thing to do. I recommend that you read the articles mentioned here (and lots more) for background. Then line up support for your ideas. Find the best liked and progressive pediatricians; let them discuss the issue with their pediatric department or medical staff. Physicians need to tell parents to stop smoking—that is their job. We help by providing education and support to the addicted smoker. To do this, we need a system that enables us to assist smokers whenever we see them.

The bulletin boards in the waiting room area of the emergency room, admission office, and parents' waiting room are natural education laboratories. Use glamorous cigarette ads, surrounded by medical facts, to catch parents' eyes. Make available simple brochures on the serious negative impact of passive smoking.

Respiratory therapists should also be activists in the community. Give talks at parent-teacher meetings and women's clubs or set up a booth at the county fair. Teach everyone you can that smoking in the presence of a child is abuse. It's the truth!

A survey of Maine pediatricians found that 91% of the respondents advised smoking parents to quit. Most spent 5 minutes discussing the risks of passive smoking. However, only 46% felt confident in advising parents how to quit.[21] Murray and Morrison, cited above, have contributed a lot to our understanding of increased morbidity related to passive smoking. They report a decrease in parental smoking in the presence of the child when parents have been advised to quit or at least refrain from smoking in the presence of the child. They also report a decrease in asthma severity in the child when this happens. They say that parents do listen to advice.[22]

The American Association for Respiratory Care recently announced Peak Performance USA, a national asthma awareness public service campaign. This program is designed to educate the three million American children with asthma and their families. A component of this program should be information on the detrimental effect of environmental tobacco smoke. Teach the children and their family members that environmental tobacco smoke includes hydrogen cyanide, a poison that attacks respiratory enzymes; nitrogen dioxide, an irritating gas; benzopyrene and tars, cancer-causing substances; nicotine; and about 4,000 other chemicals.[23] Your asthma education program should include information about the known risks of smoking, especially the health hazards to infants and children.

Today's American smoker is more likely to come from a lower socioeconomic group.[24] So how can we help them stop smoking if we cannot be re-

imbursed for this service? One thing that may help convince administrators to support our efforts is dollar savings: Work with your medical records and finance department to find out how much care for asthmatic children is written off as bad debt. If young parents without insurance are using your hospital for their children's care, it may cost you. If you put the problem in these terms, your administrator may see the value of teaching preventive care, even at no charge. The asthmatic child will be the winner, with less frequent visits to the emergency room and hospital and less frequent attacks of asthma. Let's work on it. It's the 90s thing to do!

Louise Nett RN RRT
Research Associate
PSL Center for Health Science Education
Denver, Colorado

Reprints: Louise Nett RN RRT, PSL Center for Health Science Education, 1719 E 19th Ave, Denver CO 80218.

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Measuring Total Pulmonary Compliance with a Volume-Controlled Infant Resuscitator: A Bench Study

Niranjan Kissoon MD, Gamal Monem MD, Suzanne Murphy PhD, Norman Tiffin RRT, and Timothy Frewen MD

BACKGROUND: The NeoV_{O_2}R, a volume-controlled resuscitator (VCR), can deliver an adjustable and known tidal volume. We conducted this study to determine whether this VCR can be used to measure total pulmonary compliance. DESCRIPTION OF DEVICE: The VCR studied consists of a hand-held bulb connected to an inflatable bellows inside a graduated cylinder. When the hand bulb is squeezed, the bellows expands and displaces an equal volume of oxygen or air from the graduated cylinder to a maximum of the preset volume. EVALUATION METHODS: Pressure-volume measurements comparing the VCR to an 80-mL syringe were made using 8 test lungs. Each test lung was inflated with several volumes (from 5 to 80 mL) via a 4-way stopcock using the VCR or the syringe in random order, and 2 plateau pressures were recorded. In a second experiment, 8 different test lungs were used to compare pressure-volume measurements using the VCR against the pneumotachygraph. Several pressure and volume recordings were made using each test lung, as the ventilator and VCR settings were adjusted to generate a compliance curve over a wide range of volumes and pressures. Regression lines and *t* tests were used to analyze the data. EVALUATION RESULTS: VCR vs syringe—the slopes and intercepts of regression lines were not significantly different ($p > 0.5$). VCR vs pneumotachygraph—the slopes of regression lines were not different ($0.5 > p > 0.2$). Mean compliances between the VCR and the syringe and pneumotachygraph were not significantly different ($p = 0.93$ and $p = 0.84$, respectively). CONCLUSION: Bench measurements suggest that the VCR studied may be useful as a simple bedside method of measuring total pulmonary compliance. Further testing in infants is warranted. [Respir Care 1993;38(9):985-992.]

Dr Kissoon is Director, Pediatric Intensive Care Unit, Wolfson Children's Hospital, and Associate Professor, University of Florida School of Medicine, Jacksonville, Florida. Dr Monem is Fellow in Critical Care Medicine, Wolfson Children's Hospital and University of Florida School of Medicine, Jacksonville, Florida. Dr Murphy is Assistant Director of Clinical Investigation, Nemours Children's Clinic, Jacksonville, Florida. Dr Frewen is Director, Pediatric Intensive Care Unit, Children's Hospital of Western Ontario and Professor of Pediatrics, University of Western Ontario, London, Ontario, Canada. Mr Tiffin was Research Coordinator, Pediatric Intensive Care Unit, Children's Hospital of Western Ontario, London, Ontario, Canada when this work was done.

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Reprints: Niranjan Kissoon MD, Wolfson Children's Hospital, Suite 203, Howard Building, 820 Prudential Dr, Jacksonville FL 32207.

Background

Barotrauma is the most common iatrogenic complication in neonatal intensive care units and occurs as a result of numerous factors, including overzealous ventilation.[1,2] Overzealous ventilation may be due to failure to recognize and respond to improvement in pulmonary compliance in neonates with lung disease.[3] Early determination of subtle changes in pulmonary compliance would, therefore, be of benefit to the clinician in the management of these infants. However, this technology is not readily available at the bedside.

The lack of a simple means of measurement is partly due to the fact that the currently available inflation and occlusion techniques of measuring pulmonary compliance rely on sophisticated and expensive equipment such as pneumotachygraphs, pressure transducers, and computers. Recently a vol-

ume-controlled resuscitator, the NeoV$_{O_2}$R (VCR)* has been shown to be advantageous in the provision of manual ventilation to infants and neonates when compared to self-inflating resusci-tators.[4] The VCR delivers fixed tidal volumes ranging from 10 to 100 mL, and the resulting pressure changes can be measured easily using an in-line manometer. We sought to determine whether results of bench testing would support the potential use of the VCR for measuring total pulmonary compliance at the bedside.

Methods & Materials

Design of Volume-Controlled Resuscitator

The VCR studied is essentially a bellows system (Figs. 1 & 2), consisting of a hand bulb (D) connected to a bellows (G) situated within a graduated cylinder (K). This cylinder is connected to the patient's circuit (I), which is in series with the exhalation valve (J). The desired volume of gas is obtained by releasing the lock-nut (C) and moving the adjustable piston (E) inside the graduated cylinder. This device has been described previously.[4]

This VCR is able to maintain PEEP via a PEEP valve located just distal to the exhalation valve (J, ie, away from the patient). The weight of the VCR with an accompanying circuit is 312 g (11 oz), dead space is < 1 mL when connected to the endotracheal tube, and system compliance is 0.125 mL/cm H_2O.

Inspiration. The fresh gas flow (A, Fig. 2A) fills the entire pressure chamber (M) through one-way valves (H), and excess gas flows through the one-way outflow valve (F) to the atmosphere. When the hand bulb is squeezed, it causes inflation of the bellows (G), which displaces the desired gas volume through the circuit (I) to the patient's lungs and simultaneously closes the exhalation valve (J).

Expiration. The bulb (D, Fig. 2B) is released and reinflates, causing the bellows (G) to deflate. The subatmospheric pressure created in the empty chamber draws fresh gas through the one-way valves (H) and simultaneously causes the exhalation valve (J) to open. The one-way outflow valve (F) closes to prevent entrainment of room air into the chamber. If the flow filling the chamber is insufficient, the pressure equalizing bellows (B) inflates in response to the subatmospheric pressure within the cylinder (N) and allows the chamber to fill, without slowing the recovery of the hand bulb. One-way valves (L) in the patient's circuit prevent rebreathing.

Calculation of Pulmonary Compliance

The compliance of the pulmonary system is based on volume-pressure relationships and equals the change in volume divided by the change in

*Suppliers are identified in the Product Sources section at the end of the text.

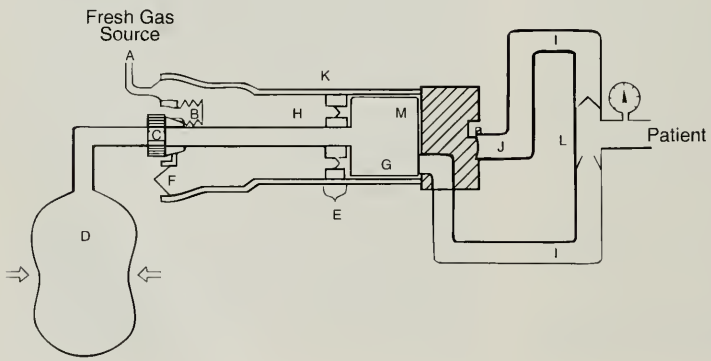

Fig. 1. The NeoV$_{O_2}$R (VCR), A = fresh gas source; B = pressure-equalizing bellows; C = lock nut; D = hand bulb; E = adjustable piston inside of graduated cylinder; F = 1-way outflow valve to atmosphere; G = bellows; H = 1-way valve; I = circuit; J = exhalation valve; K = graduated cylinder; L = 1-way valves to prevent rebreathing; M = pressure chamber; N = cylinder.

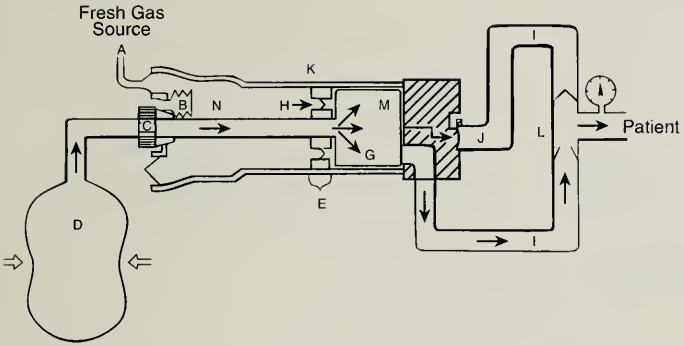

Fig. 2A. The VCR during inspiration. Arrows indicate direction of gas flow. See Figure 1 legend for key.

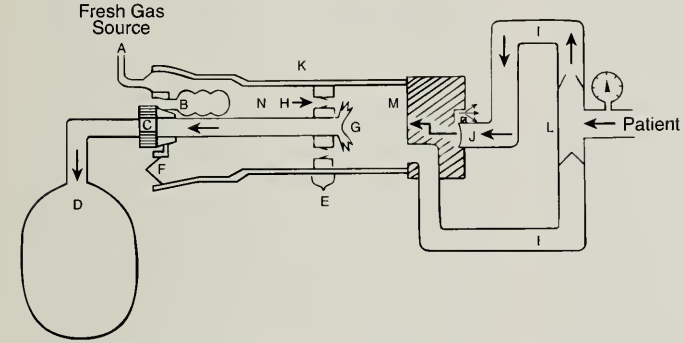

Fig 2B. The VCR during expiration. Arrows indicate direction of gas flow. See Figure 1 legend for key.

pressure. We were interested primarily in the pressure within the test lung as representing 'alveolar' pressure. This variable can also be quantitated by measuring test-lung pressure at its opening ('mouth' pressure) under no-flow conditions. We have found this easy to achieve clinically even in infants who are spontaneously breathing because their Hering-Breuer reflex is strong, and at occlusion volumes above end-expiratory volumes, the respiratory muscles will normally be relaxed.

We measured changes in pressure when a plateau of > 2 seconds was obtained with varying changes in inflation volume. For this VCR, the volume used was that 'dialed into' the device while the pressure was recorded by the manometer. In comparison, when the ventilator was used, flow was measured by the pneumotachygraph attached to a low-range pressure transducer, and the output was electronically integrated to provide the volume signal.

Study 1

We initially tested the VCR against an 80-mL syringe, using 8 test lungs. These lungs were constructed of varying lengths of corrugated tubing and thin-walled latex balloons to provide different lung compliances. The test lungs were connected to 1/8-in PVC corrugated tubing attached to a pressure monitor and printer and a strip-chart recorder. The VCR and syringe were connected to test lungs via a custom-made, 4-way stopcock as shown in Figure 3. Each lung was secured to reduce motion artifacts and potential changes in compliance.

Prior to the study, each lung was leak-tested by ensuring a non-decaying positive plateau pressure

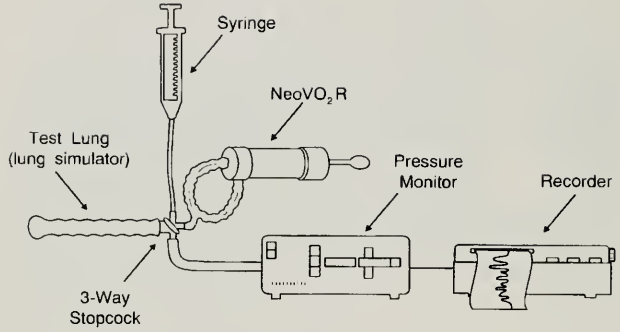

Fig. 3. Experimental setup for Study 1 with VCR and syringe.

for at least 3 seconds at 50 cm H_2O. During testing, the latex was not allowed to expand past the point where its compliance fell or remained static with increasing volume. Lungs not satisfying the above criteria were discarded. The VCR used in these experiments were leak- and performance-tested according to the manufacturer's instructions. Each test lung was then inflated with 5 to 80 mL of room air via the 4-way stopcock using the syringe or VCR in random order. The syringe or VCR was used to deliver varying volumes of air (from 5 to 80 mL) into the test lungs. The starting volume in all cases was 5 mL which was then increased in increments of 10 to 20 mL to a maximum of 80 mL. Volumes were increased and decreased in a similar manner for both the VCR and the syringe to provide 2 pressure recordings at each volume. The pressure was noted at the mouth connector of the 1/8-in PVC tubing and recorded on the strip-chart recorder (Fig. 3).

Study 2

In this bench study, we simulated the clinical scenario in which compliance testing would be undertaken in a mechanically ventilated child. The VCR was compared with the pneumotachygraph using 8 different test lungs that had been constructed and tested as outlined in Study 1. An infant ventilator was connected by way of a 4-way stopcock to a pneumotachygraph, pressure transducer, integrator and 2-channel recorder as shown in Figure 4. The test lung was connected to a pressure monitor and to the pneumotachygraph and ventilator via the 4-way stopcock using 1/8-in PVC tubing. In order to minimize dead space, the pres-

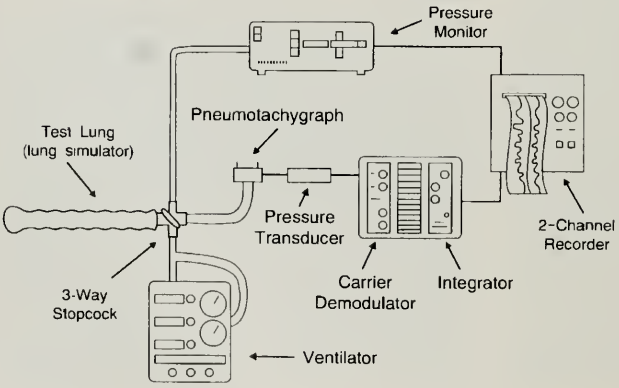

Fig. 4. Experimental setup for Study 2 with VCR and pneumotachygraph.

sure monitor was connected by the shortest length of PVC tubing possible to the 4-way stopcock at the opening of the test lung. The pneumotachygraph and pressure monitor were calibrated using a graduated syringe or water column, respectively. For compliance measurements using the VCR, the ventilator was disconnected at the 4-way stopcock and the VCR was inserted into the circuit.

Tidal volumes from the infant ventilator into the test lungs were interrupted at various points in the inspiratory cycle using the 4-way stopcock, which diverted the remaining inspiratory flow to ambient air while holding the volume in the test lung until a plateau pressure had been achieved for 2 seconds. The air was then diverted via the 4-way stopcock from the lung to the pneumotachygraph, and the exhaled volume was recorded when the lung was deflated to its natural state. Because we were interrupting the inspiratory cycle of the ventilator at random times, we were unable to use the exact volumes as in Study 1; however, volumes over a similar range were used. Several pressure and volume recordings were made using each lung as ventilator settings were adjusted to generate a compliance curve over a wide range of volumes and pressures. The plateau pressure and volume readings, using the VCR and these lungs, were obtained as described in Study 1.

Results

Regression analysis and *t* tests were used to analyze the data. Depicted in Figure 5 are the regression lines for the VCR and syringe for Study 1. Slopes and intercepts were not significantly different [$t_{(0.975,\ 112)} = 0.07$ and $t_{(0.975,\ 113)} = 0.18$; $p > 0.5$ for both]. Regression lines for the VCR and pneumotachygraph in Study 2 are shown in Figure 6. Slopes were not significantly different [$t_{(0.975,\ 183)} = 0.73$, $0.5 > p > 0.2$], but intercepts were significantly different [$t_{(0.975,\ 137)} = 3.58$, $p < 0.001$].

Mean compliances were calculated for each trial of each study. Shown in Figure 7 are the mean compliances for Study 1. There was no significant difference in mean compliance between the VCR and syringe [$t_{(0.975,\ 14)} = 0.09$, $p = 0.93$]. Similarly, a comparison of mean compliance between the VCR and pneumotachygraph (Fig. 8) revealed no statistically significant difference [$t_{(0.975,\ 14)} = 0.2$, $p = 0.84$].

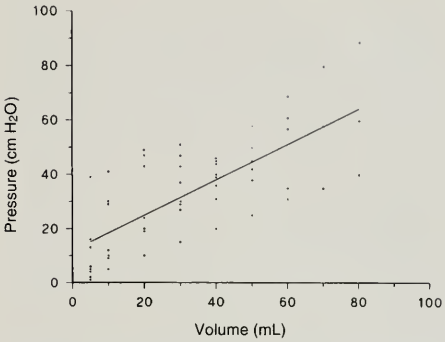

Fig. 5A. Comparison of volume and pressure regression lines for Study 1 with VCR (r = 0.76).

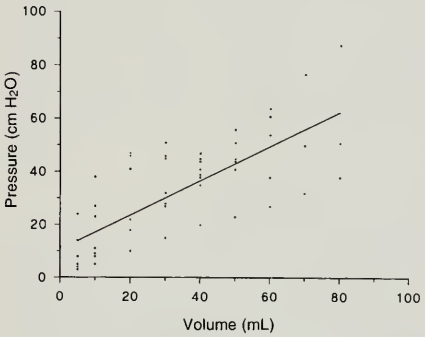

Fig. 5B. Comparison of volume and pressure regression lines for Study 1 with syringe (r = 0.75).

Discussion

The increased survival of critically ill neonates requiring mechanical ventilation has been accompanied by a high incidence of both acute and chronic complications of mechanical ventilation such as barotrauma, air leak, and bronchopulmonary dysplasia.[1] Pulmonary barotrauma may be reduced if changes in pulmonary compliance are recognized.[2,3] In addition, measurement of pulmonary compliance may be useful in intensive-care settings for titration of bronchodilators and sur-

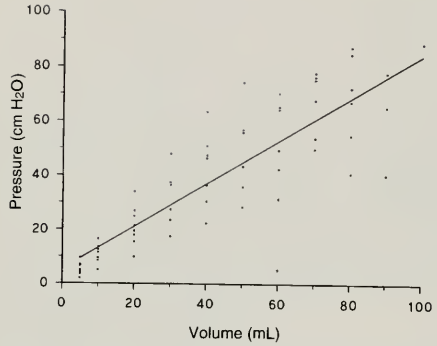

Fig. 6A. Comparison of volume and pressure regression lines for Study 2 with VCR (r = 0.87).

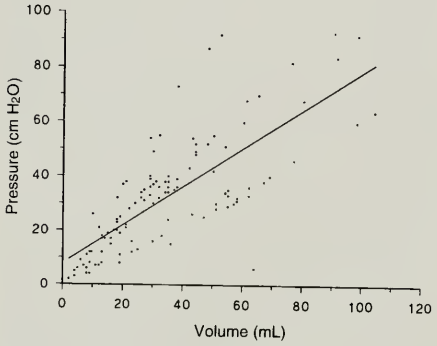

Fig. 6B. Comparison of volume and pressure regression lines for Study 2 with pneumotachygraph (r = 0.77).

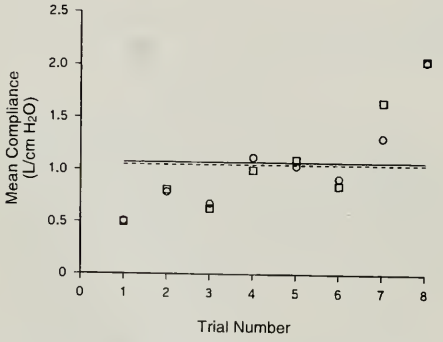

Fig. 7. Comparison of mean compliance for VCR (○---○) vs syringe (□—□) for each trial of Study 1.

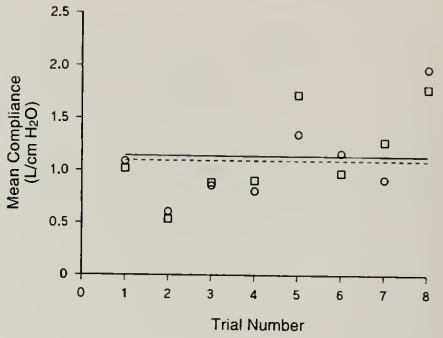

Fig. 8. Comparison of mean compliance for VCR (○---○) vs pneumotachygraph (□—□) for each trial of Study 2.

factant therapy and for successful weaning from mechanical ventilation.[5-8]

Previous investigators have stressed the prognostic value of lung compliance in predicting mortality,[9,10] development of chronic lung disease,[11] and successful weaning from extracorporeal membrane oxygenation in newborns.[12]

Despite recognition of the importance of evaluating pulmonary compliance, bedside compliance is not measured routinely. This is partly due to the fact that current methods of assessment require sophisticated and expensive equipment and are cumbersome in the congested ICU environment. We felt that the delivery of a preset volume with an in-line pressure manometer might provide a useful and simple method of measuring compliance. This method would be useful because the commonly held belief that the "educated hand" may permit the clinician to detect subtle changes in pulmonary compliance has been disputed.[13] Apart from the recognition of changing compliance due to disease processes, acute changes due to pneumothoraces and endobronchial intubation may also be detected.

The current methods of determining pulmonary compliance in ventilated infants are based on either occlusion or inflation techniques.[14] Both methods rely on sophisticated and expensive equipment, including a pneumotachygraph, pressure transducers, and computers. The occlusion technique depends on performing multiple brief occlusions at various points of the tidal breath during respiration. For meaningful data, the infant should be relaxed be-

cause changes in functional residual capacity (FRC) during the occlusion can alter the baseline volume resulting in increased variation in volume-pressure data. The inflation technique using self-inflating resuscitators may also be difficult to perform and interpret because it is technically difficult to obtain data over the range of the tidal volume of the patient.

In Study 1, we obtained similar compliance results using the VCR and syringe. This is not surprising because similar volumes were delivered by both methods. These data suggest that assessment of pulmonary compliance may be possible whenever the VCR is used during manual ventilation of an intubated patient. Our subsequent experiment (Study 2), comparing the VCR with compliance measurements using a pneumotachygraph, represents its use in a patient being ventilated in the pressure-control mode. In this instance the patient may need to be disconnected from the ventilator and manually ventilated with the VCR for compliance determination. The similarity of both slopes using the VCR and the pneumotachygraph suggest similar pressure-volume relationships and hence compliance measurements.

In Study 1, our results (Fig. 5) show similar slopes and intercepts in pressure-volume relationships for the VCR and the syringe. In Study 2, the slopes are similar (Fig. 6), but the intercepts are different. The similar slopes suggest that serial measurements using the VCR may be used to follow changes in pulmonary system compliance. The fact that a comparison of mean compliances for VCR vs syringe (Fig. 7) and VCR vs pneumotachygraph (Fig. 8) shows no significant differences also indicates that this device shows some promise.

These bench studies suggest that assessment of pulmonary compliance by VCR is feasible and may be worthwhile. However, difficulties encountered in inadequately sedated infants or in those with large leaks around the endotracheal tubes cannot be addressed by this study. Because computations of compliance and resistance rely on accurate measurements of the volume of air delivered to the airway and lungs, the presence of a large leak renders the computations inaccurate. However, this condition will be apparent to the user because of a spontaneous decay in plateau pressures associated with any substantial leak around the endotracheal tube. The removal of any patient from PEEP to measure pulmonary compliance is also of concern. The VCR is able to circumvent this problem because PEEP can be maintained by dialing in the appropriate value at the PEEP valve located at the exhalation valve.

In our experiment because the balloons used were deflated totally between measurements, the issue of end-expiratory volumes was irrelevent. Therefore, we conducted only two measurements at each volume. In the newborn, however, the end-expiratory volume is often greater than FRC and hence many pressure-volume data sets will be required to accurately determine pulmonary compliance. We are studying the application of the device to infants. These studies may further clarify the differences in methodology between clinical and bench settings. In summary, our study suggests that the VCR has the potential to provide a means of measuring total pulmonary compliance at the bedside. Further investigations in infants are warranted.

PRODUCT SOURCES

Volume-Controlled Resuscitator:
NeoV$_{O_2}$R, Neomed Inc, London, Ontario, Canada

Test-System Components:
Low-range pressure transducer, model MP45, Validyne Engineering Corp, Northridge CA.
Flow integrator, model 8815A, Hewlett Packard Co, Walton MA.
Pneumotachygraph, model 8311A, Hans Rudolph Co, Kansas City MO.
Pressure monitor, model P-7, Bird Medical Products, St Paul MN.
Strip-chart recorder, Ladd model, Princeton Medical Care, Hudson NH.

Infant Ventilator:
Ventilator: Infant Star, Infrasonics Inc, San Diego CA
Circuit: model RES81-080A, Inspiron, Baxter Inc, Deerfield IL.

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Adult Respiratory Distress Syndrome during Pregnancy: A Case Report

Berrin Ceyhan MD, Turgay Çelikel MD, and Nejat Ceyhan MD

Introduction

The adult respiratory distress syndrome (ARDS) is a well-known risk of sepsis,[1] but it is uncommon during pregnancy—the rate of occurrence being 0.1% in a recent 5-year study at a university medical center.[2] Amniotic fluid embolism, eclampsia, placental abruption, and tocolytic therapy may cause ARDS in the gravid patient.[3] The most important intervention in the therapy of ARDS is identification of the underlying abnormality and its correction when reversible.

Pregnancy predisposes women to acute pyelonephritis,[4] and urolithiasis is also known to contribute to the development of acute pyelonephritis.[5] Pyelonephritis complicated by pulmonary injury was first reported in four pregnant patients in 1984 by Cunningham et al.[6] Two other individual cases have since been reported in patients with similar histories.[7,8] In 1987, Cunningham et al[9] reported on 15 antepartum patients identified during a 7-year period, whose pregnancies were complicated by acute pyelonephritis and who developed various degrees of respiratory insufficiency. In this paper, we report a case illustrating that a pregnant woman with urolithiasis may develop acute pyelonephritis and septic shock leading to ARDS that can be treated successfully in an intensive care facility.

Dr Berrin Ceyhan and Dr Çelikel are in the Division of Pulmonary Medicine, and Dr Nejat Ceyhan is in the Department of Obstetrics and Gynecology—Marmara University Hospital, Istanbul, Turkey.

Reprints: Berrin Ceyhan MD, Marmara University Hospital, 81190 Altunizade, Istanbul, Turkey.

Case Summary

A 25-year-old woman, 20 weeks into her first pregnancy, was admitted to the obstetric service at Marmara University Hospital complaining of dysuria, lumbar pain, and inguinal pain of 5 days' duration. Physical examination was unremarkable except for finding right-sided costovertebral tenderness. Laboratory analysis revealed a white blood cell count (WBC) of 7,200/mm^3, hemoglobin of 9 g/dL, and pyuria. Other laboratory values were normal. Ultrasonography of the abdomen revealed two calculi (2 × 2 cm) and pelvicalyceal ectasia in the right kidney. Fetal development corresponded to 20-weeks gestational age. A diagnosis of acute pyelonephritis was made; and after a urine specimen had been obtained for culture, intravenous cefotaxime therapy was instituted empirically.

On Hospital Day 3, at 8 AM, the patient developed headache with nausea. The systolic blood pressure dropped to 60 mm Hg and the heart rate increased to 120 beats/min, although body temperature remained normal. Laboratory tests showed a WBC of 17,000/mm^3, hemoglobin decreased to 7.6 g/dL, and serum creatinine concentration of 1.7 mg/dL. The central venous pressure (CVP) was 0 cm H_2O.

Amikacin sulfate 500 mg bid, cefaperazone 2 g bid, dopamine 4 $\mu g \cdot min^{-1} \cdot kg^{-1}$, and fluid replacement were started, with the diagnosis of septic shock due to acute pyelonephritis; cefotaxime was discontinued. Over a 3-hour period, 3,200 mL of isotonic saline and 5% dextrose solutions were administered, followed by 2 units of blood. Systolic blood pressure increased to 90 mm Hg within 2 hours of fluid resuscitation. At 6 PM, respiratory

distress developed and the respiratory rate increased to 36 breaths/min. Chest radiography showed bilateral alveolar infiltrates suggesting ARDS. At that time, the CVP increased to 10 cm H_2O, and arterial blood gas-pH values on oxygen at 10 L/min via face mask were P_{aO_2} 69 torr, P_{aCO_2} 48 torr, and pH 7.2. The patient's lung injury score was 2.3 (mild to moderate).[10]

The patient was immediately intubated with an 8-mm endotracheal tube, and mechanical ventilation (MV) was initiated with a BEAR 5 volume-cycled ventilator. Initial settings were V_T 12 mL/kg, mandatory rate 20/min, PEEP 10 cm H_2O, and F_{IO_2} 0.75. During MV, the patient was initially paralyzed with a total dose of 6 mg pancuronium bromide and sedated with a total dose of 10 mg diazepam and a total dose of 2 mg flunitrazepam. After 15 min of MV, the blood gas-pH values were P_{aO_2} 282 torr, P_{aCO_2} 20 torr, pH 7.46.

On Hospital Day 4 (MV Day 2), PEEP of 10 cm H_2O and F_{IO_2} 0.50 were needed to keep the P_{aO_2} about 70 torr. The dopamine infusion was continued for 36 hours after the patient became hypotensive; the dose was tapered to 3 $\mu g \cdot min^{-1} \cdot kg^{-1}$ with improving blood pressure. On Hospital Day 5 (MV Day 3), pentothal 350 mg I.V. was given during a change of the endotracheal tube to a 7.5-mm nasotracheal tube. There was no growth in the urine culture. MV with 10 cm H_2O PEEP was continued through MV Day 4; on MV Day 5, PEEP was decreased to 5 cm H_2O. The patient was extubated on MV Day 6. Pulmonary infiltrates gradually improved, and antibiotic therapy continued unchanged for 10 further days.

On Hospital Day 17, in her 23rd week of pregnancy, the patient was discharged on amoxicillin 2 g/day orally. Surgical removal of uroliths was not considered, even though nausea, vomiting, and pyuria recurred in the 33rd week of pregnancy. When *Escherichia coli* was cultured from the urine in the 33rd week, the patient was rehospitalized and the infection was treated with a 14-day course of tobramycin I.V., after which she was again discharged.

In the 38th week of pregnancy, the patient was admitted to the hospital in active labor with breech (footling) presentation. Arthrogryposis and agenesis of the corpus callosum were present in the infant, who died at 6 months of age because of swallowing and feeding problems. The mother's urolithiasis was managed with lithotrypsy following delivery, and she was completely normal 1 year after her episode of ARDS.

Discussion

The case reported here fits the established criteria of ARDS.[1] In reviewing our patient's clinical course, we find that acute pyelonephritis and septic shock appear to be the most likely factors predisposing to her ARDS. Reports of ARDS developing as a complication of acute pyelonephritis during pregnancy are rare in the literature. One patient with a history similar to that of our patient developed ARDS 36 hours after the initiation of antibiotic therapy.[6] She received mechanical ventilatory support for 84 hours and gave birth to a healthy infant 8 weeks later. The group who published this report later reviewed 15 antepartum patients who had respiratory insufficiency with pyelonephritis, comparing them with 30 matched control subjects.[9] A higher incidence of multisystem disorders (such as tachycardia, hemolysis, and intravascular coagulation) was found in the patients than in the controls. No clinical risk factors were identified that could be used on hospital admission to predict pulmonary injury. Pulmonary injury occurred in 15 of 750 (2%) cases of antepartum pyelonephritis.

The authors of the study[9] suggested that the syndrome probably was caused by membrane-permeability pulmonary edema, probably mediated by endotoxin-induced alveolar capillary membrane injury. All the patients had a delay, or "lag phase," of up to 48 hours between the time of hospital admission and clinically manifested evidence of respiratory insufficiency. Leukocytosis, thrombocytopenia, positive blood culture, serum creatinine > 1 mg/mL, I.V. fluid therapy, and klebsiella or proteus infections were more prominent in women with pulmonary injury. Four of the 15 patients were > 15 weeks pregnant. MV was instituted in 3 of the 15 patients for 3-5 days, and only 1 of these 3 survived; the other 2 died, presumably from endotoxemia. By comparison, our patient had leukocytosis and high creatinine levels, with ARDS developing 2 days after admission. She survived without any other organ dysfunction.

In 119 patients with pyelonephritis only, Towers et al[11] observed pulmonary injury associated with antepartum pyelonephritis in 11, for an incidence of 9%. Highly predictive of pulmonary injury in these patients—when they were > 20 weeks pregnant—were a maternal heart rate > 110 beats/min and a fever > 103°F 12-24 hours before the occurrence of respiratory symptoms, fluid overload, the use of tocolytic agents, or the use of ampicillin or cephalosporins only as the first line of treatment. The authors suggested that these risk factors should be closely monitored, especially if the patient is > 20 weeks pregnant. Only 2 of the 119 patients studied had renal stones; one of these was in the pulmonary-injury group. By comparison, our patient's heart rate was 120 beats/min, she was 20 weeks pregnant, and cefotaxime was administered initially.

Kilpatrick and Matthay[2] reviewed all obstetric patients admitted to a university medical center ICU in the USA over a recent 5-year period. These ICU patients (n = 32) represented 0.4% of all obstetric patients in that period, and 25% of the 32 (n = 8) had acute lung injury (ie, ARDS). Thus, the incidence of ARDS among all obstetric patients in that center for the study period was 0.1%. In all the ARDS cases, the primary clinical disorder was sepsis. Two of the 8 had sepsis with pyelonephritis; one of these was ventilated, and both survived. The mortality rate among the 8 ARDS patients was 25%. This suggests that pneumonia and pyelonephritis in pregnancy should be treated aggressively, although a 1989 review[12] pointed out that maternal mortality has remained unchanged since 1955 despite advances in critical care.

In a recent paper, Collop and Sahn,[13] noting the lack of literature on the need for intensive care for critically ill obstetric patients, report on 20 such patients admitted to a medical ICU (MICU) over a 40-month period. Half of the 20 had pre-existing medical problems. Four of the mothers (20%) and 7 of the infants (35%) died. ARDS was present in all the maternal deaths. Mortality and the need for MV were not different between the 20 obstetric and 825 nonobstetric patients in the MICU during this period, but the obstetric patients, though younger (mean age, 24 years) than the nonobstetric patients (mean, 53 years), appeared to have as great a risk of dying as the general MICU population, and they had a very high infant mortality. The authors conclude that "Intensive care specialists who care for critically ill obstetric patients should be familiar with the more common complications of pregnancy and should work closely with obstetricians in their comprehensive care, in order to improve maternal and fetal outcome."

In our patient, the most likely cause of ARDS was septicemia and septic shock, to which she was predisposed by pregnancy and pyelonephritis. Relapses of pyelonephritis can occur before and just after delivery in this population.[14] The second attack of pyelonephritis occurred despite ampillicin prophylaxis.

Although the mother recovered completely, her infant had arthrogryposis and agenesis of the corpus callosum. One might ask about the possible mechanisms for fetal anomalies in this patient. Early intubation, muscle relaxation, and sedation of the mother contributed to her recovery from respiratory insufficiency. Concern over the fetal effects of the drugs used for these purposes is well founded; their use may be dangerous for the fetus. Based on the risk factors for teratogenicity assigned by the U.S. Food and Drug Administration,[15] with B signifying the least risk and D the greatest risk, the drugs we administered to our patient could be classified B: cefotaxime, amoxicillin, cefaperazone; C: amikacin, dopamine, flunitrazepam, pancuronium bromide; D: pentothal, diazepam, tobramycin.[16] Placental perfusion depends on cardiac output; hypoxemia and shock may have affected fetal tissue perfusion in our patient. Dopamine infusion was administered to increase mean arterial pressure and uterine blood flow, but in animal studies it has been confirmed that dopamine produces a decrease in uterine blood flow.[17] The lack of information regarding the effects of vasoactive drugs on uterine blood flow in critically ill patients is perhaps also a risk factor for fetal anomaly.

In the light of this case, we reviewed the literature concerning arthrogryposis and agenesis of the corpus callosum. Based on embryologic data, the deleterious effects must begin before or during the 11th to 20th weeks of gestation when the corpus callosum develops,[18] and before or during the 4th to 7th week of gestation when active limb formation ensues.[19] In one report,[20] an infant with arthrogryposis and mental retardation was delivered

by a mother with septic shock and hyperthermia in the 29th gestational week. In other papers,[21,22] a number of cases were reported in which arthrogryposis was associated with agenesis of the corpus callosum; in-utero hypotensive/ischemic insult was considered to be the pathogenesis of these anomalies. The anomalies found in the infant of our patient were most likely coincidental, as the insult occurred in the 20th week of gestation.

In summary, septic shock and the adult respiratory distress syndrome continue to be important sources of mortality in the intensive care setting. This case report demonstrates how critical care can sometimes yield a favorable outcome for a pregnant patient with ARDS.

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Occupational Asthma in the Respiratory Care Worker

W Michael Alberts MD MBA

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Introduction

Bronchial asthma is a common affliction that is estimated to afflict 10 to 15 million people in the United States. The proportion of this large population whose disease is due to occupational exposure is not known with certainty but has been estimated to be 2-5%.[1] In Japan, it has been estimated that 15% of asthma in men is due to an occupational exposure.[2] Using these figures, it can be estimated that from 200,000 to 2,250,000 individuals have occupational asthma (OA) in this country.

More is known of the prevalence of asthma in specific exposures or occupations. Prevalence figures, however, vary from 1% to nearly 100% and appear to depend on a number of factors, including the specific industrial agent, the type, source, and concentration of exposure, working conditions, industrial hygiene, engineering factors, climatic influences, economic factors, and host susceptibility.[3]

Studies have noted that approximately 4% of workers exposed to western red cedar develop asthma.[4] A similar percentage of workers exposed to volatile isocyanates develop OA.[5] Clinical symptoms of asthma reportedly occur in 10% to 45% of workers exposed to proteolytic enzymes.[6] Allergic symptoms developed in approximately 70% of flight crews dispersing irradiated sterile male screw worm flies.[7] Almost 100% of workers in the power plants along the Mississippi River eventually became sensitized to river flies.[8]

At present, over 200 agents have been reported to cause OA. This number has grown and will no doubt continue to grow rapidly as our knowledge expands and as new compounds are introduced into the workplace. Moreover, it appears that OA is rapidly becoming the most prevalent of all occupational lung diseases. In 1988, OA represented the principal occupational respiratory ailment both in terms of new claims for compensation and in total number of claims to the Worker's Compensation Board in Quebec, surpassing asbestosis and silicosis.[9]

Pathogenesis

The mechanisms involved in the development of asthma in those exposed to the responsible agent in

Dr Alberts is Associate Professor of Medicine and Public Health, Department of Internal Medicine, Division of Pulmonary, Critical Care, and Occupational Medicine, University of South Florida College of Medicine, and Chief, Division of Pulmonary and Critical Care Medicine, and Medical Director of Respiratory Care, H Lee Moffitt Cancer Center and Research Institute—Tampa, Florida.

Reprints: W Michael Alberts MD, Division of Pulmonary, Critical Care, and Occupational Medicine, 13000 Bruce B Downs Boulevard (111C), Tampa FL 33612.

the workplace are largely unknown. What is known may be divided into (1) immunologic mechanisms, (2) nonimmunologic mechanisms, and (3) unknown or unproven mechanisms.

Occupational asthma of immunologic origin is primarily mediated through specific IgE antibodies. IgG- and cell-mediated mechanisms may be important in some circumstances. Immunologically mediated occupational asthma is characterized by (1) sensitization to a specific agent (as demonstrated by skin or serologic testing) and (2) a latent period between first exposure and the onset of symptoms. The immunologic mechanism of OA pathogenesis is no different than that of the more common inhalant allergens such as the house-dust mite.

OA of nonimmunologic origin is a condition without latency, whereby injury and inflammation of the airways develop after a workplace exposure to an irritant gas, vapor, or fume and is manifested by variable airflow limitation and the presence of persistent nonspecific airways hyperresponsiveness.[3] The clinical syndrome, reactive airways dysfunction syndrome (RADS) is an example of OA that is not immunologically derived.

In a number of instances, the mechanisms involved in the development of OA are unknown or unproven. The mechanism may very well be immunologic, but confirmation remains elusive. There is a strong possibility, however, that alternate mechanisms may be operative.[3] Examples of the latter are (1) pharmacologic actions such as beta blockade or anticholinesterase activity, (2) non-immunologic mast-cell degranulation, (3) direct activation of complement, and (4) potentiation of neuropeptide activity. At present, it is not known whether any of these mechanisms is clinically important.

Epidemiology

Given this backdrop, it stretches the imagination little to envision cases of OA developing in healthcare workers in general and respiratory care workers in particular. However, the prevalence of OA in this latter group is unknown because the necessary epidemiologic studies have just not been done.

In 1989, Kern and Frumkin[10] reported a cross-sectional questionnaire study designed to test the hypothesis that work as a respiratory therapist is associated with an increased risk of developing asthma. The impetus for this study came from concern expressed by respiratory care practitioners at a local hospital in noting that 4 of the 20 members in the department had developed asthma after entering the profession. While acknowledging the potential problems imposed by their study design, the authors concluded from analyzing responses from 194 respiratory therapists and 517 controls that a previously unrecognized excess of asthma exists in respiratory therapists. (After controlling for covariates, respiratory therapy carried an odds ratio of 3.2, 95% confidence interval, CI, 1.9-5.5.) Moreover, the excess apparently develops after entry into the profession. When analysis was restricted to those who developed asthma after entering the profession, the odds ratio was 4.6, 95% CI, 2.0-10.4. That is, RTs are 4.6 times more likely to contract asthma than non-RTs; these are significantly increased odds ($p \cong 0.01$). The authors called for further research and suggested that prospective cohort studies of respiratory therapists would help both in corroborating their findings and in identifying etiologic factors. In addition, industrial hygiene evaluations of the work environment might provide clues to identify etiologic agents.

Potential Etiologies

Given the Kern and Frumkin study[10] as the best available information and accepting their conclusion that an excess of asthma occurs in this working population, what are some of the most likely (or possible) causes of OA in respiratory care practitioners? Not one of the proposed etiologies has been proven to cause excess asthma cases in the respiratory care worker, and few of the exposures are unique to this occupation. Nevertheless, although more investigation is needed, a review of the potential etiologies may be beneficial (Table 1).

Antiseptics and Disinfectants

As an integral part of their duties, respiratory care practitioners are exposed to various disinfectant solutions during the cleaning and maintenance of equipment. Exposure to these agents in heavy concentrations or in lesser amounts repeatedly over a period of time might be a possible etiology.

Table 1. Potential Causes of Occupational Asthma in Respiratory Care Practitioners

Antiseptics and disinfectants	
Glutaraldehyde	Hexachlorophene
Chloramine-T	Formaldehyde (formalin)
Chlorhexidene	Ethylene oxide
Sodium hypochlorite	
Latex gloves	
Challenge agents (Pulmonary function laboratory)	
Methacholine	Histamine
Drugs and medications	
Beta-lactams	Piperazine
Macrolides	Penicillamine
Tetracycline	Methyldopa
Hydralazine	Salbutamol
Isoniazid	Beta Agonists (?)
Pentamidine	Psyllium
Research laboratory animals	
Animal dander	Serum
Saliva	Urinary proteins
Irritant Inhalation Injury—RADS	
Glacial acetic acid	Toxic spills
Microbial Agents	
Viruses	Chlamydia
Mycoplasma	

Glutaraldehyde is a widely used disinfectant that is especially effective for cold sterilization of instruments.[11] This chemical is a known mucous-membrane irritant and has been reported to cause allergic contact dermatitis.[12] Glutaraldehyde has also been reported to cause OA in individuals involved in cleaning endoscopy equipment.[13]

Chloramine-T, a potent disinfectant, is widely used in cleaning floors and other surfaces in operating rooms and laboratories. It has also been used to disinfect aerosol delivery equipment. In 1973, Van Bork et al[14] reported OA due to chloramine-T in operating room nurses. A number of subsequent reports, including the 1981 paper by Dijkman et al,[15] have confirmed the entity and have suggested that the pathogenesis involves an IgE-mediated mechanism.

In 1989, OA was reported to develop in nurses exposed to a chlorhexidene and alcohol aerosol used to disinfect incubators.[16] This substance is a widely used disinfectant that has been a suspected skin-sensitizing agent. However, the report did not provide enough information to separate the irritant effect of chlorhexidene and its potential for sensitization. The latter mechanism might then result in allergic OA.

Household bleach (5.25% sodium hypochlorite) is used by respiratory therapists on occasion (for example, in the maintenance and cleaning of blood gas analyzers). Repeated exposure to chlorine may cause rhinorrhea, coughing, wheezing, and other respiratory problems.[11] Although generally well tolerated when used alone, reports of toxic pneumonitis and RADS have appeared when this solution is mixed with other household cleaners (such as ammonia).[17]

In 1984, Nagy and Orosz[18] reported a case of OA felt to be due to hexachlorophene. This substance is used as a powder or solution for topical disinfection. The patient, a nurse, had been working in contact with hexachlorophene for 15 years. She had had respiratory symptoms for about 10 years. Initially, her symptoms were of rhinitis, but for the 2 years prior to evaluation she had noted frequent asthma attacks.

Formalin is an aqueous solution with 37% to 50% formaldehyde by weight (10 to 15% methyl alcohol is added to prevent polymerization). Formalin is used as a disinfectant and as an embalming fluid. OSHA has estimated that 1.3 million workers are exposed to formaldehyde and that 1 in 3 of these work in healthcare professions or settings. Exposure to formaldehyde produces many symptoms, chiefly ocular and nasal irritation. This substance has been suspected of causing lower respiratory tract symptoms such as cough and wheezing. Hendrick and Lane[19] reported that two nurses working in a renal dialysis unit developed asthmatic symptoms associated with their work with formaldehyde. The exposures involved were of relatively high concentrations and the pathogenesis of these symptoms is in question. Bardana[20] has recently concluded that despite the fact that it has been suggested that formaldehyde may lead to sensitization, sufficiently well-controlled and reproducible scientific studies are not yet available that definitely establish the link between respiratory tract allergy and formaldehyde vapor.

Ethylene oxide is used to sterilize medical instruments, particularly those made of heat-labile materials. Although ethylene oxide has an odor threshold of about 700 ppm, exposure at 200 ppm may cause irritation of the eyes and upper respiratory tract. Dugue et al[21] have recently reported OA provoked by ethylene oxide in a nurse.

Latex Gloves

The AIDS epidemic and the Universal Precautions mandate have resulted in more widespread and more frequent use of gloves by healthcare workers. As a result, latex allergy manifesting as asthma is potentially important in respiratory care practitioners.

Rubber is a known cause of contact dermatitis. Additives used in the manufacturing process have usually been named as the responsible sensitizing agent. In 1979, Nutter reported the first case of contact urticaria due to natural latex.[22] Since that time, a number of case reports have been published that show immediate hypersensitivity and asthma due to latex.[23]

In 1988, Seaton et al[24] reported a case of OA due to rubber gloves. The authors believed that the patient's asthma was provoked by vapor given off by rubber gloves and they incriminated carene, which is a terpene produced naturally by plants. Other studies have suggested that the responsible substance may be natural latex proteins[25] and that exposure to the antigen occurs by inhalation possibly in association with the glove powder.[23] Marcos et al[26] recently reported the case of an operating nurse who, after 25 years of exposure to sterile rubber gloves, began to notice perennial nasal and ocular itching, sneezing, and rhinorrhea that worsened at work. Three years later she noted the onset of urticaria in areas in contact with the gloves. In addition, she noted wheezing and dyspnea following the use of sterile gloves. After intensive evaluation that included skin-prick testing, patch testing, rub testing, indirect enzyme-linked immunoabsorbent assay (ELISA), and specific bronchial challenge with latex extract, the authors concluded that the patient had OA due to latex present in surgical rubber gloves. They also suggested that latex was acting as an inhalant allergen producing symptoms through an IgE-mediated mechanism.[26]

Methacholine and Histamine

Respiratory care practitioners may be involved in pulmonary function testing in addition to their usual duties. Lundgren et al[27] recently reported two cases of increased airway responsiveness that developed in personnel conducting methacholine and histamine challenge tests. One subject developed symptoms of chest tightness and cough, especially with exercise. She had conducted methacholine challenges several times a week for more than 2 years. Symptoms improved, but she continued to require medication after she stopped conducting challenge tests. The other subject performed methacholine and histamine challenges on a daily basis for more than 2 years. She developed symptoms of asthma and required intermittent corticosteroids even after she changed jobs.

The authors of this report suggested that repeated exposure to small concentrations of histamine and/or methacholine could induce an inflammatory response in the airways and thereby increase airway responsiveness. Similar findings were reported, by letter, in two research associates performing methacholine challenges.[28] The evidence incriminating methacholine and/or histamine in these situations is circumstantial. Nevertheless, the reports serve as provocative observations.

Drugs and Medications

A number of antibiotics and other drugs have been shown to cause OA. The majority of cases have occurred in workers involved in the manufacturing process, but healthcare workers may be at risk as well. In the course of their duties, respiratory care practitioners may come in contact with the myriad of drugs and medications used in patient care.

Proteolytic enzymes (such as papain, chymopapain, bromelain, trypsin, and chymotrypsin) have been shown to cause OA. Hayes and Taylor[29] reported OA in a pediatric nurse that was caused by inhaling pancreatic extracts used in the treatment of infants with cystic fibrosis.

Drugs such as the beta-lactam antibiotics, the macrolide antibiotics,[30] tetracycline, hydralazine,[31] isoniazid, piperazine, penicillamine,[32] and methyldopa have been reported to cause OA. Once again, cases have been identified more frequently in those

involved in manufacturing. As an example, Fawcett et al[33] reported occupational asthma developing in a pharmaceutical worker involved in the production of salbutamol (albuterol). Detailed investigation showed that a glycyl compound, an intermediate chemical powder prepared during manufacture, was the etiologic agent rather than the final compound.

Respiratory care practitioners are exposed to aerosolized bronchodilators repeatedly during the course of the day. It has been suggested that long-term use of the beta agonists might lead to tachyphylaxis to the effect of these medications. Theoretically, this might occur in the respiratory care practitioner who is frequently passively exposed. However, this phenomenon would likely be operative only in those with pre-existing asthma or bronchial hyperresponsiveness and would therefore be unlikely to cause asthma de novo.

Another possible etiology would be the additives or contaminants in commercially prepared beta agonists.[10] Repeated exposures may place those exposed at risk of developing OA. For example, in the past, bisulfites were added to nebulized bronchodilators as preservatives. If this mechanism is operative, it is undoubtedly rare today.

OA due to psyllium is perhaps more peripherally related to respiratory therapy than to nursing, but it does exist. Ground husks and seeds of the psyllium plant are used as a bulk laxative preparation. OA due to psyllium is more commonly described in those involved in manufacturing.[34] OA has been described in healthcare personnel handling psyllium.[35,36] Malo et al[37] investigated the prevalence of OA and immunologic sensitization to psyllium among health personnel in chronic care hospitals. They found that the prevalence of sensitization was between 5% and 12% and that the prevalence of OA was 4%; they concluded that psyllium posed a significant risk to the respiratory health of personnel in chronic care hospitals.

Aerosolized pentamidine has been approved for prevention of *Pneumocystis carinii* in patients with acquired immunodeficiency virus infection. Occupational exposure to aersolized pentamidine in medical personnel has raised two concerns: passive exposure to the drug and possible transmission of respiratory pathogens.[38] Montgomery et al[38] have concluded that the amount of passive pentamidine is very small compared to the doses received by the patients in whom long-term adverse effects are few. Patients receiving aerosolized pentamidine may note a metallic or bitter taste, cough (38%), and perhaps bronchospasm (15%).[39] The reduction in airflows may be reversed or prevented by beta-agonist aerosol treatment.[40] Montgomery and coworkers[38] have concluded that the greater risk is probably transmission of respiratory pathogens (tuberculosis).

No cases of asthma due to occupational exposure to pentamidine have been reported. In one study of patients receiving aerosolized pentamidine for 10-14 months, no increase in bronchial reactivity as measured by methacholine could be detected.[41] However, anecdotal reports of a reversible decrease in diffusing capacity and ocular irritation have been published.[42] Respiratory therapists in our department have experienced chest tightness after delivering prophylactic treatments with pentamidine. A detailed study of respiratory therapists working in five pentamidine aerosol clinics in San Francisco is now being conducted.[40]

Animal Products

Respiratory care practitioners working in research who are regularly exposed to animals are at risk of developing sensitization to animal dander, saliva, serum, and urinary proteins. Prevalence rates for animal allergy range from 11% to 15% in the United States.[43] The most common manifestation is allergic rhinoconjunctivitis.[44] Between 25% and 50% of these individuals will develop symptoms of bronchial asthma.[43] Atopic individuals are more likely to develop asthma from sensitization to animal products.

Irritant Inhalation Injury

Asthma may develop as the result of an exposure to high concentrations of a respiratory irritant. OA acquired in this manner has been termed RADS. RADS develops without a latent period and is distinguished by persistent nonspecific airway hyperresponsiveness. The relevant exposure is characteristically acute, singular, and extreme in nature.[45]

Many exposures have been reported to cause RADS. Kern has recently described an outbreak of RADS after a spill of glacial acetic acid.[46] Fifty-six

hospital employees were exposed to 100% acetic acid when a technician in the nuclear medicine laboratory attempted to lift a 1-gallon bottle of the chemical. Of the 51 employees subsequently studied, 1 of 30 with medium exposure and 3 of 14 with high exposure developed symptoms and met the diagnostic criteria for RADS.[46] Respiratory care practitioners are at risk for similar exposures, and may thus develop nonallergic OA in this manner.

Microbial Agents

Perhaps the most intriguing potential etiology of OA in respiratory care practitioners is the exposure to microbial agents. The respiratory care practitioner is exposed to patients with viral upper and lower respiratory tract infections, bacterial bronchitis and pneumonia, tuberculosis, and fungal diseases on a regular, if not daily, basis.

It has been well accepted that viral and *Mycoplasma pneumoniae* respiratory tract infections may precipitate an acute asthma attack in those with pre-existing disease. Additionally, it can be shown that non-specific bronchial responsiveness increases after a viral infection and may remain increased for a number of weeks.[47]

It has been suggested that viral infections may actually serve as the pathogenetic event that results in the asthmatic state. Many patients date the onset of their asthma to a particularly severe respiratory infection. If this theory proves to be correct, the apparent increased risk of developing OA in respiratory care practitioners could very well be explained by exposure to patients with respiratory tract infections.

Recently, investigations have shed light on how viral infections may give rise to wheezing, bronchial hyperresponsiveness, and, possibly, asthma (Table 2[47]).[48] Viral infections probably do not induce direct changes in the contractile properties of airway smooth muscle. However, infection with respiratory viruses may influence bronchial smooth muscle by a number of mechanisms, including (1) production of virus-specific IgE antibodies that may sensitize basophils and mast cells, (2) amplified granulocyte-dependent inflammation, (3) enhanced mediator release that can result in bronchial obstruction directly or by producing airways inflammation, and (4) epithelial damage.

Table 2. Possible Mechanisms of Virus-Induced Asthma*

Production of virus-specific IgE antibodies
Amplified granulocyte-dependent inflammation
Enhanced mediator release
Sensitization of afferent vagal sensory fibers
Epithelial damage
Exposure of irritant receptors
Loss of relaxing factors and degrading enzymes
Facilitation of allergen presentation
Beta-adrenergic blockade

*Adapted from Reference 47.

Airway epithelial damage and disruption is a common finding in viral infections. This damage may lead to (1) enhanced cholinergic reflex bronchospasm due to exposure of rapidly adapting sensory fibers located beneath the basement membrane and (2) the loss of production of such factors as neutral endopeptidase, which degrades bronchospastic neurokinins and bronchial smooth-muscle relaxing factors. Bacterial infections of the respiratory tract apparently do not share the propensity to exacerbate asthma. An exception is bacterial infection of the paranasal sinuses.

In 1991, Hahn et al[49] reported a relationship between chlamydial pneumonia and asthma in adults. The authors found that patients with serologically confirmed *Chlamydia pneumoniae* infection were much more likely to subsequently develop asthma than were seronegative patients (antibody titers less than 1:16). Almost 30% of seropositive patients were subsequently diagnosed as having asthmatic bronchitis as opposed to 7% of seronegative patients during a 6-month follow-up. The authors state that chronic infection or re-exposure to *C pneumoniae* could trigger an immunopathologic process in the lungs, perhaps involving epithelial damage and mediator release or delayed hypersensitivity to chlamydial protein antigens, to cause the chronic airway inflammation characteristic of asthma. Although the study was not specifically designed to test a hypothesis about wheezing or asthma, the identified association is intriguing.[50]

In Conclusion

Occupational asthma has been identified in healthcare workers in general and in respiratory care workers in particular. However, the incidence,

prevalence, and etiologies have been insufficiently investigated at present. Given the nature of their employment, respiratory care workers are exposed to the entire gamut of the patient population and the myriad of support services. As a result, these workers are exposed to any number of possible agents that may give rise to asthma; therefore, further investigation of these substances and those heretofore unrecognized agents is necessary to minimize or eliminate the risk of respiratory care workers' developing occupational asthma.

Until the necessary information becomes available, it seems prudent to adopt reasonable preventive measures. For example, adequate ventilation of work areas should be assured, work practices should be evaluated and, if possible, be re-designed to minimize potential exposures, and the use of personal respiratory protection devices (eg, face shields) should be considered. Perhaps, most importantly, the respiratory care practitioner should become aware of and informed about potential hazards encountered in the work place.

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A Strategy for Reducing Costs Associated with Pulse Oximetry in Noncritical Care Areas

Michael J Mahlmeister MS RCP RRT, James B Fink MS RCP RRT, and Neal H Cohen MD MPH MS

Introduction

Since its introduction into clinical practice in the early 1980s, the pulse oximeter has gained widespread application. The availability of an accurate, portable, inexpensive device for continuous, noninvasive assessment of arterial oxygen saturation (S_{pO_2}) has provided clinicians with a tool to guide their management of an ever-increasing variety of clinical situations. Figure 1 is a list of some of the many applications of pulse oximetry—extensive yet not all-inclusive because the list continues to grow each year.

The pulse oximeter was initially used to assure the adequacy of oxygenation in the operating room, where some signs and symptoms of hypoxemia may be absent or obscured by drapes. The value of the pulse oximeter in surgery and postoperative recovery rooms as a safety device to reduce the incidence of hypoxemia and its sequelae became immediately apparent. A number of reports from the literature in the early 1980s described the value of pulse oximetry monitoring as an early warning sign of hypoxemia and promoted its use as a tool to reduce morbidity and mortality in anesthesia practice. Several malpractice insurance companies reduced their premiums for those anesthesiologists who routinely incorporated pulse oximetry monitoring into their practice.[1] In 1986, the Department of Anesthesia of Harvard Medical School published standards for patient monitoring during anesthesia, which included continuous pulse oximetry,[2] and the American Society of Anesthesiologists recommended that S_{pO_2} monitoring become a standard of care in the operating and recovery room.[3]

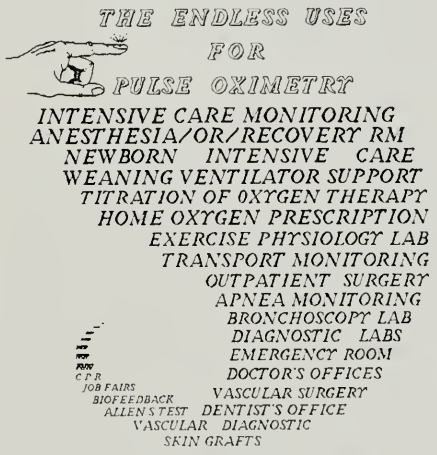

Fig. 1. Some of the many uses of pulse oximetry.

During the late 1980s, the use of pulse oximetry expanded, particularly in the areas outside of the operating room, including critical care units and cardiopulmonary diagnostic units.[4,5] S_{pO_2} mon-

Mr Mahlmeister is Education Director, and Dr Cohen is Professor of Anesthesia and Medicine, Vice Chairman, Department of Anesthesia, and Director, Intensive Care Unit and Respiratory Care Service—The Medical Center at the University of California, San Francisco. Mr Fink was Technical Director, Respiratory Care Service, The Medical Center at the University of California, San Francisco when the study was performed; he is now Program Analyst, Hines Veterans Administration Hospital, Chicago, Illinois.

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Reprints: Michael Mahlmeister MS RRT, UCSF Respiratory Care Service, Room M-69, 505 Parnassus Ave, San Francisco CA 94143-0206.

itoring became routine in the critical care unit to titrate oxygen therapy and ventilatory support and to warn of hypoxemic episodes. Although the value of pulse oximetry in noncritical care settings was not well defined, many practitioners chose to adopt its use in other clinical settings, creating a dramatic increase in demand. The proliferation of applications is apparent from the recommendations of one author that S_{pO_2} monitoring be considered as "a fifth vital sign,"[6] joining blood pressure, heart rate, respiratory rate, and temperature as a routine nursing measurement. However, although many articles promoted the clinical value of S_{pO_2} monitoring, there was a paucity of literature describing how best to apply the limited resources for patients cared for outside of the operating room and critical care settings. Should all patients on oxygen therapy be monitored by pulse oximetry, only patients with cardiopulmonary disorders, or only those patients on an F_{IO_2} of "X" or greater? Which patients should receive continuous S_{pO_2} monitoring versus intermittent spot checks? A 1988 editorial in RESPIRATORY CARE[7] raised these very questions, and called for our profession to "work to establish pulse oximeter standards or guidelines." The AARC Clinical Practice Guideline on Pulse Oximetry, published in RESPIRATORY CARE in December 1991, was an answer to this call for standards.[8]

Pulse Oximetry and the RCP

The role of the respiratory care practitioner (RCP) in guiding the adoption and appropriate use of pulse oximetry has grown hand-in-hand with the technology, particularly with respect to its use as a monitor of the appropriateness of oxygen therapy. Because RCPs maintained responsibility for oxygen therapy in most hospitals in the early 1980s, it followed that this new technology for monitoring the adequacy of oxygenation should also fall within the domain of the RCP. Smoker et al[9] reported on the effective use of pulse oximetry by RCPs as part of an oxygen therapy administration protocol, leading to improved patient care and a reduction in arterial blood gas analysis. King and Simon[10] studied the application of pulse oximetry in weaning patients from oxygen therapy, concluding that S_{pO_2} monitoring reduced both the number of days of oxygen use and institutional costs associated with arterial blood gases. Kochansky[11] described the use of ear oximetry during rest, position change, and exercise in determining prescriptions for home oxygen. The 1991 AARC Clinical Practice Guideline on oxygen therapy[12] attests to the important role of the RCP in oxygen therapy, and the necessity of incorporating S_{pO_2} monitoring into that role.

Background

As the clinical applications for pulse oximetry grew in the 1980s, like many other institutions The Medical Center at the University of California, San Francisco (UCSF) experienced a significant increase in requests for pulse oximetry in noncritical-care settings. Figure 2 illustrates the dramatic rise in orders for pulse oximetry between 1985 and 1988 at our 560-bed teaching institution, a 375% increase between 1986 and 1988 in orders for continuous pulse oximetry in other than critical care units. This led to serious difficulties in meeting physician demands for continuous monitoring and for frequent and/or immediate 'spot-check' S_{pO_2} monitoring, despite a 400% increase in oximeter rentals.

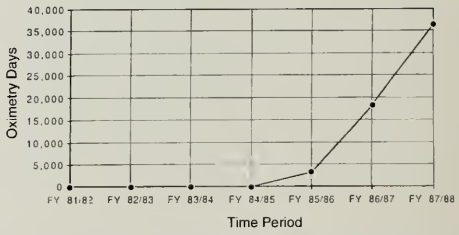

Fig. 2. The rise in pulse oximetry use at the University of California-San Francisco Medical Center. Between Fiscal Year 85-86 and the close of 87-88, oximetry-days rose from fewer than 5,000 to more than 35,000.

The concerns regarding appropriate S_{pO_2} monitoring were compounded at UCSF because in 1985 the nursing service had assumed responsibility for all oxygen therapy in noncritical care areas,[13] while Respiratory Care Service (RCS) retained responsibility for all pulse oximetry monitoring for these patients. This 'sharing' of patient care responsibilities clearly represented an ineffective use of human resources, magnified by growing physician demands for pulse oximetry monitoring. Our de-

partment initially attempted to meet increased demands for S_{pO_2} monitoring through a variety of strategies, including: (1) physician education programs, with specific clinical criteria for use of continuous vs spot check monitoring outlined in memos to medical staff; (2) increased purchase and rental of pulse oximeters, targeted for use in noncritical care areas; and (3) RCS staffing changes aimed at designating staff available for immediate response to requests for S_{pO_2} spot checks.

Despite these strategies, the department was forced to create a waiting list of patients for whom continuous pulse oximetry had been ordered. Physicians were informed when their requests had been entered on a waiting list, and the order was carried out as soon as an oximeter became available. This policy led to considerable expenditure of RCS staff resources each day, in attempts to have pulse oximetry discontinued from one patient so that the oximeter could be made available for another patient on the waiting list. Physicians complained about the lack of an adequate number of oximeters and the poor response time of RCS in complying with new orders. Nurses and, to a lesser extent, physicians were switching oximeters between patients, bypassing our department in an effort to avoid having a patient placed on a waiting list. Paradoxically, during rounds, our staff frequently found that "continuous" oximeters were often turned off to avoid unnecessary alarms caused by patient movement. Nurses were intermittently turning the oximeter on to obtain an S_{pO_2} reading. In essence, the patient was utilizing an expensive, continuous pulse oximeter for periodic spot checks.

RCS and nursing staff frustrations grew in proportion to the ineffective attempts to control the utilization of pulse oximetry and the conflicts that arose among RCPs, physicians, and nurses. Concurrently, as Figure 3 illustrates, institutional costs associated with oximeter rentals and the purchase of disposable probes rose sharply, particularly from May to September 1988. The existing strategies to control the use of pulse oximetry were inadequate, and other strategies were needed to better match this limited resource with patient need and at the same time control costs and reduce staff stress. The program below outlines our institution's efforts to address this increase in pulse oximeter use in a cost-effective manner that best matched limited human and equipment resources with patient need.

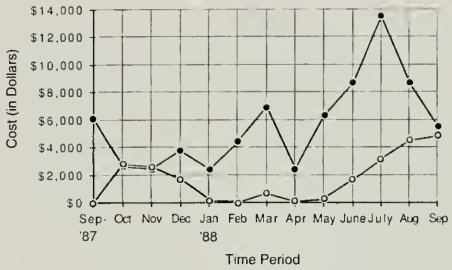

Fig. 3. Increased costs associated with the rise in the number of orders for continuous pulse oximetry on patients in noncritical-care areas. Supply costs were for the purchase of disposable probes. Supplies –●–; rentals –○–.

The Program

The first step in developing a solution was to clearly identify the problem. Although we had data on the overall growth of pulse oximetry use in the institution, specific use patterns were less clear. In early 1988, RCS implemented a 5-month data collection project in an effort to answer three questions: (1) What nursing units were high users of pulse oximetry? (2) Which services tended to order pulse oximetry? (3) What were the most common reasons for pulse oximetry orders? The results of this data collection demonstrated that orders for continuous pulse oximetry ranged from 1-20 orders/month/nursing unit. Data also identified the medical specialties responsible for the majority of orders and showed that most requests for pulse oximetry were made to assess the adequacy of oxygenation for patients already receiving oxygen therapy.

With this information available, our department initiated the formation of a multidisciplinary task force to explore strategies to address pulse oximeter utilization. Those represented on the task force included RCS (technical and medical director), Nursing Administration, Hospital Administration, and Materiels Management. Following a review of institutional use patterns and cost analysis, the task force developed a plan to implement a pilot project aimed at altering usage patterns. Two nursing units with high use were selected for the pilot project. Each of these units was

provided with two Nellcor N-10 Pulse Oximeters (Nellcor Inc, Hayward CA). RCS conducted 30-minute in-service programs for the nursing staff, covering concepts of pulse oximetry, operation of the N-10, the purpose of the pilot project, and the required documentation. The medical director drafted a letter to all attending and house-staff physicians describing the pilot project and explaining why spot checks are adequate for most patients at an ordered frequency not to exceed every 4 hours. An interval of 4 hours was selected to correspond with routine vital sign measurements as ordered on medical-surgical patients.

For the nursing units involved in the pilot project, all physician-ordered spot checks were performed by the nursing staff, without the need to contact RCS. In addition, nurses were encouraged to obtain S_{pO_2} readings on patients whenever they felt the need to do so, without a physician's order. Nursing staff were instructed to make all physicians aware of the availability of spot-check oximetry in an effort to decrease physician orders for continuous oximetry. RCS retained responsibility for all continuous pulse oximeter monitoring.

Figure 4 illustrates the data collection sheet used by the nursing staff during the pilot project. Full results of the 2-month project are presented in Table 1. A total of 897 spot checks were documented by nursing personnel. Complete information was available for 833 cases. For 818 of 833 cases (98%), nurses indicated that they felt the spot check was valuable. Of 753 spot checks, 229 resulted in a change in therapy (Table 1): F_{IO_2} change, suctioning, aerosol therapy and/or repositioning.

Under Reason indicate whether check initiated by RN or MD and circle one of the following.

1. Routine check
2. Change in Clinical Status
3. Change in Ozygen Therapy
4. One time check of ozygenation

Date/ Time	Pt. Name/RM #	O2 Flow/ Device	SpO2	Reason for check	WasCheck Valuable ?	Change in Therapy Result?
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
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				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO
				Ordered by: RN MD 1 2 3 4	YES NO	YES NO

Fig. 4. Data collection sheet used during the 2-month pilot project.

Table 1. Results of the 2-Month Pilot Project for Pulse Oximetry Conducted on Two Nursing Units at UCSF (10L & 7L)*

Number of Spot Checks?	
Total (10L, 476; 7L, 421)	897
Initiated by RN	228
Ordered by MD	390
Patients already on O_2	507
Reason for Check?	
Routine	302
Change in clinical status	28
Change in oxygen therapy	76
One-time check of oxygenation	188
Check Valuable?	
Yes	818
No	15
Change in Therapy?	
Yes	229
No	526

*Note: Not all fields in the form were completed for each spot check, consequently the sum of reported results does not equal the total number of spot checks reported.

During a pilot project follow-up meeting, nurses voiced almost unanimous support for the spot-check program. They felt better able to assess the patient without the logistic or temporal delays associated with the previous system. No significant concern was expressed about the nursing time commitment. Rather, nurses felt that S_{pO_2} monitoring fit quite easily into their normal nursing routine of taking vital signs. Most importantly, they expressed satisfaction with the fact that they now had a tool at their immediate disposal to assess the adequacy of oxygen therapy for which they held responsibility.

Based upon evaluation of the pilot project data, and the overwhelmingly positive response from nursing, the task force implemented the training program and distributed Nellcor N-10 pulse oximeters to all noncritical care nursing units over a 3-month period. An inservice program was instituted for these nursing units—including a videotape and outline that was available for nurses unable to attend the program and for new employees and temporary staff.

Subsequent to the placement of portable spot-check oximeters on all nursing units, a marked decrease occurred in orders for continuous pulse oximetry. Figure 5 illustrates data on continuous pulse oximeter use for the 12 months preceding and 12 months following implementation of the program. There was a 67% decrease in orders for continuous monitoring on the general floors; within 3 months following completion of the spot-check program, RCS was able to eliminate the waiting list for oximeters.

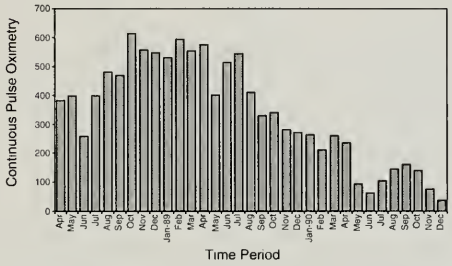
Continuous Pulse Oximetry
700
600
500
400
300
200
100
0
Apr May Jun Jul Aug Sep Oct Nov Dec Jan-89 Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec Jan-90 Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec
Time Period

Fig. 5. Data on the volume of orders for continuous pulse oximetry for the 12-month period prior to and following implementation of the spot-check program. Note the steady decrease in orders for continuous oximetry beginning with July 1989, the first month of full implementation of the program.

Financial Impact

The change instituted in pulse oximeter monitoring had important financial implications. Figure 6 illustrates the period of increasing use from September 1987-88. During this time, patient charges far exceeded costs; however, the actual reimbursement (approximately 30% of charges) made the provision of oximetry a break-even endeavor. Costs associated with the expanding use of oximeters on the general patient floors included rental costs averaging $6.60/day. Costs for disposable probes averaged $15/day. RCP time was equivalent to 40 minutes/day/patient, including time to find an available unit or to request rental of an oximeter, initial setup time, and 10 minutes of therapist time for checks and documentation every 8 hours. Average labor costs were $15.00/unit/day. This analysis does not include the overtime costs associated with the unexpected increase in patient care demands.

Table 2 illustrates the institution's cost savings associated with the implementation of this pro-

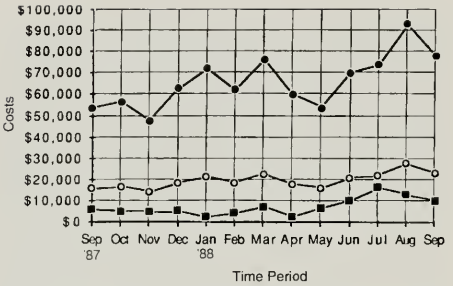

Fig. 6. Patient charges for pulse oximetry increased during 1987-88 well in excess of costs. However, due to the patient population, reimbursement rate of $0.30/$1.00 charged made pulse oximetry a break-even service for the department and institution. Patient charges —●—; reimbursement at 30% – ○ –; costs —■—.

gram. During the 12 months preceding the new program, from July 1988 through June 1989, the RCS experienced 6,250 continuous oximeter days on the general floors at a total cost of $228,750, requiring 2.34 RCP full-time equivalents (FTEs). These data do not take into account the number of patients who were placed on waiting lists due to the unavailability of rental units and who did not have the monitoring available to them. July through December 1989 was the transition period during which the program was fully implemented. During the following 12 months (January through December, 1990), oximeter days were reduced to 1,803, FTEs for this program to 0.68 FTEs, and total costs to $65,989, amounting to a 12-month savings of $162,760.20. These savings have been sustained to the present time, with a total of 1,242 oximeter days for fiscal year 1992-1993. RCS continues to provide continuous S_{pO_2} monitoring in specific situations, such as for monitoring patients who have undergone mandible or neck surgery.

Whereas this reduction in oximetry days was associated with reduction in direct patient charges for oximetry, the FTE savings with this program were used to provide services that were much less supply-cost intensive, improving the cost-revenue ratio for the departments. With the reduction in demand on the patient-care units, response time was improved for patients requiring continuous monitoring. It is difficult to quantify the time savings by staff who no longer had to chase after scarce equipment, while explaining to the medical team why service had been delayed and assuring them that the monitor would be made available as soon as possible. Likewise, it is impossible to quantify the savings associated with patients receiving more timely and appropriate service.

Discussion

By placing responsibility for spot check pulse oximeter monitoring in the hands of nurses, the program substantially improved monitoring response time and reduced costs for both patients and the institution. A review of 94 patients on 4 nursing units in June 1993 showed that 91/94 patients (97%) had received a total of 550 S_{pO_2} spot checks during the previous 48 hours, averaging 6 checks/patient. This included all 25 patients on oxygen therapy at the time data were collected. In 547/550 checks (99.4%) conditions were noted under which readings were obtained (eg, room air, cannula at 2 L/min). Patients receiving oxygen therapy usually had orders to titrate F_{IO_2} or liter flow of oxygen to maintain a desired minimum S_{pO_2}, allowing the nurse to adjust or discontinue oxygen as necessary. Of note, only 6 of 94 patients had written physician orders requesting a specific frequency of S_{pO_2} monitoring. The majority of S_{pO_2} measurements were voluntarily initiated by nursing staff, as a convenient and available 'routine' vital sign rather than as a specifically ordered task. Indeed, it appears that the nurses are using S_{pO_2} monitoring as "the fifth vital sign."

It is not clear whether this program has served the needs of all patients requiring oximetry on the general patient care floors. There is the possibility that hypoxemic episodes are missed because continuous monitoring is not being used. However, based on our experience with continuous monitoring on the floors, we found that the monitors were turned off much of the time, and alarms were frequently inactivated. The spot-check transfer program was integrated into our quality assurance monitoring, with no evidence that patient care had been compromised as a result of the program. However, as Bowton et al point out,[14] questions remain regarding the value of continuous S_{pO_2} monitoring as a tool for documenting episodes of hy-

Table 2. Costs Savings associated with Implementation of the Nursing Spot-Check Oximetry Program at USCF—Full Implementation Occurred in July 1989

Month	Oximeter Days	Total Costs	FTE	Rentals	Disposable Sensors*	Labor
April	382	$ 13,981.20	1.72	$ 2,521.20	$ 5,730.00	$ 5,730.00
May	398	$ 14,566.80	1.79	$ 2,626.80	$ 5,970.00	$ 5,970.00
June	258	$ 9,442.80	1.16	$ 1,702.80	$ 3,870.00	$ 3,870.00
July	399	$ 14,603.40	1.80	$ 2,633.40	$ 5,985.00	$ 5,985.00
August	481	$ 17,604.60	2.16	$ 3,174.60	$ 7,215.00	$ 7,215.00
September	470	$ 17,202.00	2.12	$ 3,102.00	$ 7,050.00	$ 7,050.00
October	615	$ 22,509.00	2.77	$ 4,059.00	$ 9,225.00	$ 9,225.00
November	559	$ 20,459.40	2.52	$ 3,689.40	$ 8,385.00	$ 8,385.00
December	548	$ 20,056.80	2.47	$ 3,616.80	$ 8,220.00	$ 8,220.00
January '89	533	$ 19,507.80	2.40	$ 3,517.80	$ 7,995.00	$ 7,995.00
February	595	$ 21,777.00	2.68	$ 3,927.00	$ 8,925.00	$ 8,925.00
March	555	$ 20,313.00	2.50	$ 3,663.00	$ 8,325.00	$ 8,325.00
April	576	$ 21,081.60	2.59	$ 3,801.60	$ 8,640.00	$ 8,640.00
May	402	$ 14,713.20	1.81	$ 2,653.20	$ 6,030.00	$ 6,030.00
June	517	$ 18,922.20	2.33	$ 3,412.20	$ 7,755.00	$ 7,755.00
July	545	$ 19,947.00	2.45	$ 3,597.00	$ 8,175.00	$ 8,175.00
August	412	$ 15,079.20	1.85	$ 2,719.20	$ 6,180.00	$ 6,180.00
September	331	$ 12,114.60	1.49	$ 2,184.60	$ 4,965.00	$ 4,965.00
October	343	$ 12,553.80	1.54	$ 2,263.80	$ 5,145.00	$ 5,145.00
November	283	$ 10,357.80	1.27	$ 1,867.80	$ 4,245.00	$ 4,245.00
December	272	$ 9,955.20	1.22	$ 1,795.20	$ 4,080.00	$ 4,080.00
January '90	265	$ 9,699.00	1.19	$ 1,749.00	$ 3,975.00	$ 3,975.00
February	213	$ 7,795.80	0.96	$ 1,405.80	$ 3,195.00	$ 3,195.00
March	262	$ 9,589.20	1.18	$ 1,729.20	$ 3,930.00	$ 3,930.00
April	236	$ 8,637.60	1.06	$ 1,557.60	$ 3,540.00	$ 3,540.00
May	93	$ 3,403.80	0.42	$ 613.80	$ 1,395.00	$ 1,395.00
June	64	$ 2,342.40	0.29	$ 422.40	$ 960.00	$ 960.00
July	106	$ 3,879.60	0.48	$ 699.60	$ 1,590.00	$ 1,590.00
August	146	$ 5,343.60	0.66	$ 963.60	$ 2,190.00	$ 2,190.00
September	163	$ 5,965.80	0.73	$ 1,075.80	$ 2,445.00	$ 2,445.00
October	141	$ 5,160.60	0.63	$ 930.60	$ 2,115.00	$ 2,115.00
November	76	$ 2,781.60	0.34	$ 501.60	$ 1,140.00	$ 1,140.00
December	38	$ 1,390.80	0.17	$ 250.80	$ 570.00	$ 570.00
July '88–June '89	6,250	$228,750.00	2.34	$41,250.00	$93,750.00	$93,750.00
January–December '90	1,803	$ 65,989.80	0.68	$11,899.80	$27,045.00	$27,045.00
Difference	4,447	$162,760.20	1.67	$29,350.20	$66,705.00	$66,705.00

*Base cost for sensors was $15.00 each; base cost for labor/procedure was $15.00.

poxemia leading to clinical intervention or its impact on morbidity and mortality.

We did not assess the program's impact on requests for arterial puncture and blood gas analysis. Aside from the cost implications, we believe that any decrease in ABG frequency also reduces caregiver risk of exposure to blood-borne pathogens such as AIDS and/or hepatitis.

Conclusions

Our department implemented a program that allowed for convenient, timely availability of S_{pO_2} spot-check monitoring. We recommend that other institutions explore alternatives to respiratory-care-provided S_{pO_2} monitoring in noncritical-care areas. Figure 7 illustrates the results of the spot-check transfer program, implemented in July 1992 at Mt Zion Medical Center, a UCSF affiliate hospital.

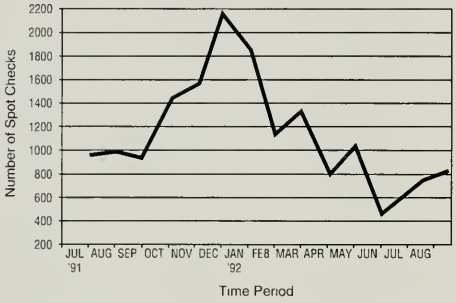

Fig. 7. Results of the S_{pO_2} spot-check transfer program implemented at Mt Zion Medical Center of UCSF in February-March 1992.

For UCSF, the decision to have spot-check pulse oximetry monitoring provided by the nursing staff seemed a logical step because they were already responsible for oxygen therapy for their patients. This strategy allowed RCS to reallocate staff to areas demanding more complex skills, an important consideration during these times of dwindling resources and budgetary constraints.

When a program such as this is instituted, it is important that Respiratory Care personnel be involved in the training of nursing personnel. A 1993 survey of 47 nursing programs in California[15] demonstrated that the mean time spent in laboratory instruction for pulse oximetry was 0.2 hours, with 21 schools indicating no laboratory time. It is clear from this survey that in 1993, graduate nurses are entering the profession with little training in pulse oximetry, in spite of its universal use in healthcare. Nurses need to appreciate the theory and application of pulse oximetry, as well as the many limitations inherent in this tool for monitoring oxygenation.[16] The RCP represents the best resource for designing and implementing a training program to successfully achieve these goals. In our institution, we were able to facilitate this transition to nursing secondary to a shared history of cooperative efforts to enhance patient care delivery in the most cost-effective manner for our institution. We encourage other departments to work collaboratively with nursing and medical staff to facilitate appropriate use of institutional resources in a manner that best meets the clinical needs and financial goals of the institution.

ACKNOWLEDGMENTS

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The Length of Educational Preparation and Academic Awards for Future Respiratory Care Practitioners: A Delphi Study

F Herbert Douce MS RPFT RRT and Deborah L Cullen EdD RRT

Introduction

Since publication of "The Future Educational Needs of Respiratory Care Practitioners: A Delphi Study" in January 1992,[1] there has been a continuing interest in the future educational preparation of healthcare providers. Influential policy organizations, such as The Pew Charitable Trust and The Kellogg Foundation, have asserted that healthcare reform can be effected through reforms of health practitioner education.[2,3] The American Association for Respiratory Care (AARC) hosted an educational consensus conference in October 1992.[4,5] Using a consensus-building normative group process, the conferees reasoned a future curriculum and educational system for respiratory care.[6,7] Important to this effort was the acknowledgment that external trends such as advancing technology and the demography of population and disease greatly influence our professional practice and education. A broadened scope of respiratory care practice and lengthened educational preparation for the future may result.

Concurrent with publication of the Delphi report[1] and based upon the knowledge, skills, attributes, and characteristics previously delineated through the Delphi process, we continued the process to determine whether 75% or more of the experts could agree on the duration and academic award for respiratory care education in the future. The purpose of this paper is to report the results of additional Delphi rounds focused on the questions of future curriculum length and academic awards for respiratory care education.

Mr Douce is Assistant Professor and Director, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio. Dr Cullen is Associate Professor and Chair, Department of Cardiopulmonary Science, School of Allied Health Science, Associate Professor of Anesthesia, School of Medicine, Indiana University, Indianapolis, Indiana.

Methodology

We used the Delphi method to conduct this study. The goal of the Delphi method is to obtain agreement among a group of experts through a series of surveys with repeated feedback. Questions in an area of concern are presented in one or more rounds to a panel of experts whose responses remain anonymous throughout the inquiry. Repeated questioning of each participant via questionnaire does not allow the individuals to interact directly with one another. The participants receive information and preliminary results between questionnaire rounds.

For the questions related to curriculum length and academic award, we surveyed, for three rounds, the same experts who participated in identifying the competencies of respiratory care practitioners. Because we considered the judgments of clinicians, managers, educators, and physicians equally important and legitimate and to reduce domination by any single constituency, these four primary groups of experts participated in similar proportions.

The selection process and distribution of the experts have been described previously in this journal.[1] The 1989-1990 AARC Education Committee requested nominations of 8 clinical practice experts from each of the 4 clinical specialty sections of the

AARC and 24 nominations for educator experts from the AARC Education Section. The educator experts encompassed accredited technician and therapist programs reflecting both traditional and nontraditional curricula and included each of four types of institutional sponsors, including proprietary schools, vocational/technical schools, community/junior colleges, and 4-year colleges and universities. The committee requested nominations of 12 management experts from the AARC Management Section to include small, medium, and large acute care facilities and university and children's hospitals, 8 additional managers from nonacute care settings, and 6 additional managers from the AARC Clinical Practice Sections. The National Association of Medical Directors of Respiratory Care (NAMDRC), the Board of Medical Advisors (BOMA) of the AARC, the National Board for Respiratory Care (NBRC), and the Joint Review Committee for Respiratory Therapy Education (JRCRTE) nominated physician experts. These 90 experts included 25 clinicians, 24 managers, 22 educators, and 19 physicians. Between the completion of the initial round and the mailing of the second round, 2 participants had ended their involvement in respiratory care and 2 participants had moved, left no forwarding address, and could not be located. Therefore, the panel was reduced to 86 experts for Rounds 2 and 3. These 86 experts included 24 clinicians, 23 managers, 20 educators, and 19 physicians.

The initial round of inquiry on the questions of curriculum length and academic award occurred in June 1989 and coincided with the fourth round of the previous study, which determined future competencies. We only included questions on entry-level curriculum length and award in the first round; we tabulated responses, identified the median response for curriculum length, and computed percentages for awards.

The second round of surveying began in October 1991; we contacted nonrespondents in January and again in February 1992. Each mailing in the second round included the competencies identified previously by these experts, each participant's individual responses, and the median of the group responses to the initial round of questioning on curriculum length. Written comments provided by the participants accompanied the response data. For this round, we defined a year in terms of academic semesters and quarters. (That is, an academic year consists of two 15-16 week semesters totaling approximately 30 semester-credit hours with each semester-credit hour encompassing a total of 15 lecture/discussion clock hours, 35 laboratory clock hours, or 90 clinical clock hours. We also defined an academic year as three 10-11 week quarters totaling approximately 45 quarter-credit hours, with each quarter hour encompassing 10 lecture/discussion clock hours, 25 laboratory clock hours, or 60 clinical clock hours.) We directed participants to consider the information we provided and to specify the suitable length and academic award (ie, degree or certificate) for entry-level and advanced-level respiratory care practitioners in the future. For length, we provided participants a continuum of 0-4 years, and for award the choices were no award, certificate of study, associate's, bachelor's, and master's degrees.

In March 1992, we surveyed the 86 experts for a third time on questions of curriculum length and academic award; we repeated the mailing to nonrespondents in April, and again in June 1992. Each mailing in this round included each participant's individual responses made in the second round and the median of the group responses to the second round of questioning on length and award. We directed participants to consider the information we provided and to specify the suitable curriculum length and academic award for entry-level and advanced-level respiratory care practitioners in the future. We concluded data collection in July 1992.

Because responses of curriculum length were on an interval scale, we tabulated responses and computed the mean, standard deviation, and variance; we then identified the median response in each round. We defined response agreement as > 75% of the experts' indicating identical responses. To determine stability of responses for program length, we compared means by a one-way analysis of variance; we tested for equality of variance, and we computed percentage of change between rounds. We defined response stability as no significant difference between two consecutive means, no significant difference between two consecutive variances,

and < a 15% change between two consecutive rounds.

Because responses of academic award were on an ordinal scale, we tabulated responses and computed percentages; we defined response agreement as > 75% of the respondents indicating identical answers. We compared the two most common responses between rounds by χ^2, and we defined response stability as no significant difference among frequencies. In all cases, we considered $p < 0.05$ as a statistically significant difference.

Results

Of the 90 experts, 76 (84%) participated in the initial Delphi round on the questions on curriculum length and academic award. Of the 86 continuing participants, 70 (81%) responded in Round 2 and 66 (77%) responded in Round 3. The overall participation rate was 81%. The numbers of experts who participated from each group are shown in Table 1.

For all three Delphi rounds, the median response for future entry-level programs was 2.0 academic years. More than 75% of the respondents failed to provide a single identical response, although more than 75% did consistently indicate 2 years or longer. These results are presented in Figure 1. For the three rounds, the mean (SD) lengths in units of academic years for future entry-level programs were 1.99 (0.68), 1.92 (0.47), and 1.88 (0.42). The differences among means ($p = 0.484$) and between the variances ($p = 0.20$) of Rounds 2 and 3 were not statistically significant. The change between means of the latter two rounds was less than 6%. More than 75% of our experts agreed on, and there was

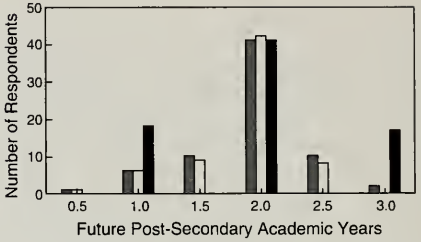

Fig. 1. Responses to questions related to future length of entry-level education. Round 1 ■; Round 2 ▩; Round 3 □.

response stability for, the length of entry-level educational preparation.

For academic award for entry level, 73% of the respondents indicated an associate's degree in Rounds 1 & 3, and 71% indicated an associate's degree in Round 2; all other responses were a certificate of completion. χ^2 analysis indicated that the differences among the frequencies were not statistically significant ($p = 0.85$). Although there was response stability on the award for entry-level educational preparation, fewer than 75% of our experts agreed on a single suitable award. These results are presented in Figure 2.

For the two Delphi rounds that asked for curriculum lengths in units of academic years for future advanced-level programs, the median response was 3.5 academic years. For Rounds 2 & 3, more than 75% of the respondents predicted programs longer than 2.5 and 3.0 years, respectively. These results are shown in Figure 3. For the two rounds, the mean (SD) academic years were 3.11 (0.99) and 3.29 (0.77). The change between means of the

Table 1. The Distribution of Experts in the Second Delphi Study, by Major Groups for Each Round

	Expert Categories				
	Clinicians	Educators	Managers	Physicians	Total
Round 1					
Mailed	25	22	24	19	90
Returned	21	18	20	17	76 (83%)
Round 2					
Mailed	24	20	23	19	86
Returned	17	19	19	15	70 (81%)
Round 3					
Mailed	24	20	23	19	86
Returned	14	17	18	17	66 (77%)

Fig. 2. Responses to questions related to future entry-level academic awards. Round 2 ■; Round 3 □.

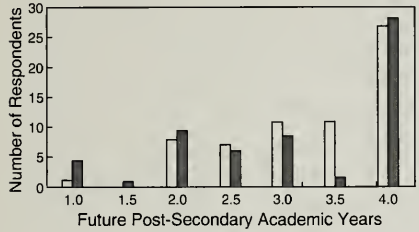

Fig. 3. Responses to questions related to future length of advanced-level education. Round 2 ■; Round 3 □.

latter two rounds was less than 6%. The difference between means was not statistically significant ($p = 0.227$); the difference between variances was statistically significant ($p = 0.02$). Fewer than 75% of our experts agreed on suitable length for advanced-level preparation; their responses remained unstable.

For academic award for advanced level, 61% of the respondents indicated a bachelor's degree in the second round and 62% indicated a bachelor's degree in the third round; almost all other responses suggested an associate's degree. These results are presented in Figure 4. χ^2 analysis for bachelor's and associate's degrees indicated that the differences among the frequencies between rounds were not statistically significant ($p = 0.50$). Although there was response stability on the award for advanced-level educational preparation, fewer than 75% of our experts agreed on a single suitable award.

Discussion

The experts had identified entry-level and advanced-level competencies of future respiratory care practitioners in the previous study.[1] These experts have suggested that two levels of respiratory care education would be suitable to develop the competencies required in the future, and that significant change may occur from what are commonly known now as 1-year entry-level certificate programs. Although our experts appear uncertain about advanced-level programs, their responses did not appear to sanction the status quo.

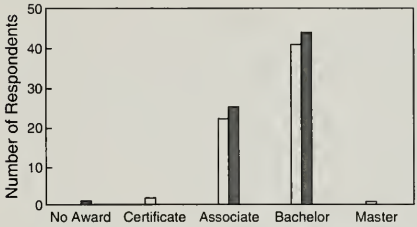

Fig. 4. Responses to questions related to future advanced-level academic awards. Round 2 ■; Round 3 □.

Because the sponsorship of respiratory care educational programs has shifted from hospitals to academic institutions, we have attempted to quantify program lengths in terms of academic years instead of calendar months. We believe that specifying the curriculum length in terms of academic years is a more appropriate measure of the duration of post-secondary education.

The results also imply that entry-level and advanced-level postsecondary educational program length and the academic recognition awarded for respiratory care educational program completion will continue to evolve. The evolution began in 1957 with the required minimum length of respiratory care education of 1 calendar year for technicians; it was extended to 18 months, referred to as 2 academic years, in 1967.[8,9] Existing technician programs were recognized as therapist programs in 1972, with required minimum curricula of 20 months.[10] In 1972, technician programs were required to last a minimum of 10 months.[11] These minimum lengths remained for 15 years, until the 1986 Essentials ended specific curriculum-length requirements in 1987.[12] With deregulation of curriculum length and the focus of accreditation on successful outcomes, program length may continue to increase to be responsive to the needs of communities of interest; increasing program length may

be necessary if the competencies of the future become reality.

Through the Delphi process, the experts have predicted a length of 2 postsecondary academic years to prepare entry-level practitioners appropriately, similar to what was required for technician programs in 1967. Our analysis of 165 technician program listings in the 1992 Allied Health Education Directory revealed a range of 8-24 months, and a mean (SD) curriculum length of 13.5 (3.0) months.[13] This average length in calendar months would be equivalent to 1.5 academic years. Our results imply that entry-level education may be prolonged further in the future. Although more than 75% of the experts failed to agree on the type of academic award for completion of entry-level preparation, as the duration of entry-level education increases, program completion may be rewarded more often by an associate's degree than by certificate.

According to Joint Review Committee for Respiratory Therapy Education data, from 1979 through 1991 the number and percentage of technician programs with lengths longer than 12 calendar months increased from 25 and 13% in 1979 to 48 and 26% in 1991. Over the course of these 13 years, the number and percentage of technician programs with extended curriculum length almost doubled.[14] The results of this Delphi study indicate that this trend may continue.

Consistent also with the results of this Delphi study, the AARC Educational Consensus Conference participants concluded that our future educational system will require an associate's degree as the minimum to enter practice.[7] Other studies of curriculum length and award have presented similar findings. In the 1984 study of the predictive validity of the 1977 Essentials, Scanlan found that above average performance on the 1983 NBRC entry-level examinations was associated with program lengths of longer than 18 months and with program sponsorships by junior/community colleges or 4-year colleges/universities.[15] In 1984, Douce and Wiezalis surveyed all 351 program directors of accredited programs on their perspectives toward the proposed 1986 Essentials. Two hundred eighteen (62%) program directors responded, and 55% of the respondents agreed or strongly agreed that the Essentials should require a 2-year minimum curriculum length, a minimum of 62 semester hours of college credit, and an associate's degree, for entry level.[16]

Through the Delphi process, the experts have not yet agreed on a specific length or award for advanced-level preparation. The results of each round predicted a longer length, culminating in a length longer than 3 postsecondary academic years to prepare advanced-level practitioners appropriately. An analysis of 262 therapist program listings in the 1992 Allied Health Education Directory revealed a range of 8-57 months and a mean (SD) length of 22.5 (6.4) months.[13] These lengths in calendar months are equivalent to 2.5 academic years, indicating that just extending advanced-level education further also may continue.

Although more than 75% of the experts failed to agree on the type of academic award for completion of advanced-level preparation, more than 60% did predict a bachelor's degree. Because only 38 of 263 (14%) therapist programs report awarding bachelor's degrees in the 1993 Allied Health Education Directory, our results may indicate future growth in the number and proportion of bachelor's degree therapist programs. Depending upon the prerogative of each sponsoring educational institution, both bachelor's or associate's degrees will continue to be awarded for advanced-level preparation.

We have reported the results of three Delphi rounds on the questions on curriculum length and academic award for respiratory care education. Participants determined that 2 or more years of education may be necessary for future entry into practice and that an associate's degree may be the academic award granted with entry-level preparation. For advanced-level preparation, students may find it necessary to attend programs for 3.5 years or longer as projected by the panelists. As previously noted, the trend toward increasing the length of educational preparation is consistent with the results of our study. Furthermore, our results complement those of the AARC Educational Consensus Conference that looks toward more traditional educational preparation of practitioners by awarding degrees appropriate to the length of postsecondary education.

Limitations of the Study

Readers should carefully consider the limitations when evaluating the results of any Delphi study.

Although we involved four major constituencies of respiratory care experts and although our panel of experts was large, not all points of view in respiratory care may be represented. Another limitation of the Delphi technique is that the validity of the predictive power of the Delphi technique for forecasting may be questionable because the proof can only be established with the passage of time. What our experts think will happen in the future may be influenced by issues unidentified by the experts. How much students are willing to pay for respiratory care education and how much employers are willing to pay graduates may determine the future curriculum length and academic award for respiratory care education.

ACKNOWLEDGMENTS

We are grateful to the 86 experts who participated and who enabled us to continue the Delphi process.

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Advanced Cardiac Life Support Update: Uses of Drugs

Hugh S Mathewson MD and Dan Conyers BA RRT

Introduction

Hospital-based respiratory care practitioners are frequently involved in cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC).[1-7] Their role as active participants was recognized at the 1992 National Conference on CPR and ECC, where representatives of the American Association for Respiratory Care maintained a close liaison with the primary sponsors. In this conference, new research data and reports of prospective clinical studies were reviewed and recommendations written for inclusion in life support manuals. The incorporation of advanced cardiac life support (ACLS) in the curricula of respiratory care (RC) programs and the inclusion of resuscitative procedures in the content matrix of the National Board for Respiratory Care place responsibilities on the profession to maintain a high level of expertise, both in training and in clinical performance.

Dr Mathewson is Professor Emeritus of Anesthesiology and Professor of Respiratory Care Education, and Mr Conyers is Assistant Director of Respiratory Therapy Services—University of Kansas Medical Center, Kansas City, Kansas.

Reprints: Hugh S Mathewson MD, Professor Emeritus of Anesthesiology, Professor of Respiratory Care Education, University of Kansas Medical Center, #5040-D, 39th & Rainbow, Kansas City KS 66103.

In this brief review, we summarize the current status of drug therapy in CPR/ECC, as set forth by the 1992 National Conference. The specifics are contained in the CPR issue of the *Journal of the American Medical Association* (JAMA, October 28, 1992).[8] The guidelines and recommendations published in this group of articles constitute an authoritative consensus, to which the student of RC can be referred in preparing for qualifying examinations.The reader may find that following the ACLS algorithms as he or she reads this Capsule increases understanding of the sequencing and timing of drug interventions.

The new guidelines are conservative in tone; the term "standards" is no longer used. They attempt, insofar as possible, to address effectiveness and appropriateness of CPR based on sound scientific evidence. Most of the previously established guidelines (1985 and earlier) are unaltered or have minor changes. In interpreting the revisions in drug protocols, the reader must understand the classification of therapeutic interventions shown in Table 1. The recommended major changes that pertain to drug administration are summarized in Table 2.

Several general guidelines for drug administration during CPR are emphasized. If peripheral venous access is utilized, intravenous (I.V.) medications should be given by rapid bolus injection, followed by a 20-mL bolus of I.V. fluid and elevation of the extremity.[9] Higher doses of drugs in larger volumes are necessary if

Table 1. Classification of Therapeutic Interventions in CPR and ECC: A System of Classifying Recommendations Based on the Strength of the Supporting Scientific Evidence*

Class I	A therapeutic option that is usually indicated, always acceptable, and considered useful and effective
Class II	A therapeutic option that is acceptable, is of uncertain efficacy, and may be controversial
Class IIa	A therapeutic option for which the weight of evidence is in favor of its usefulness and efficacy
Class IIb	A therapeutic option that is not well established by evidence but may be helpful and probably is not harmful
Class III	A therapeutic option that is inappropriate, is without scientific supporting data, and may be harmful

*Adapted from Reference 1 (Page 2174).

Table 2. Drug Administration in ACLS* and PCLS: Recommended Changes from Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care[8]

Epinephrine
ACLS—for repeat doses, high-dose epinephrine (up to 0.1 mg/kg) is given a Class IIb status.
PCLS—repeat dose of epinephrine at 0.1 to 0.2 mg/kg is Class IIa.
Sodium Bicarbonate
In hyperkalemia—Class I
In bicarbonate-responsive metabolic acidosis—Class IIa
On return of spontaneous circulation after long arrest—Class IIb
In hypoxic lactic acidosis—Class III
Calcium Salts
In calcium-channel blocker overdose—Class IIa
In known ionized hypocalcemia—Class IIa
Otherwise—Class III
Adenosine
For paroxysmal supraventricular tachycardia in adults—Class I (drug of choice)
Thrombolytic Agents
For acute myocardial infarction—Class I

*ACLS = adult advanced cardiac life support; PCLS = pediatric advanced life support.

they are instilled endotracheally. This applies mainly to ephinephrine, lidocaine, and atropine. It is recommended that 2 to 2.5 times the usual I.V. dose be given, diluted in 10 mL of normal saline or distilled water.[10] If I.V. access is unavailable, the interosseous route can be employed; this is especially applicable in infants and small children. The interosseous dose is ordinarily the same as the I.V. dose, but the ephinephrine dose may need to be increased. Glucose-containing fluids are not recommended for resuscitative efforts (Class III) because of possible deleterious effects on the brain, although 5% dextrose in distilled water (D_5W) is stated to be acceptable.

Antiarrhythmic Drugs

Lidocaine remains the drug of choice for treatment of ventricular ectopic rhythms (Class I). Although the incidence of ventricular fibrillation (VF) following acute myocardial infarction (MI) is reduced by prophylactic I.V. infusion of lidocaine, the margin of safety is narrow, and use of the drug routinely in patients suspected of having MI is probably unwarranted.[11] In cases of MI without ventricular premature beats, the recommendation is Class IIb. Lidocaine is also indicated for wide-complex paroxysmal supraventricular tachycardia (PSVT), or for tachycardias of uncertain type.

Procainamide is recommended for suppression of ventricular ectopy when lidocaine is contraindicated or has failed. The indications for procainamide are virtually identical to those cited for lidocaine, but it is not the drug of choice and is listed as Class IIa. Its chief hazard is severe hypotension, which is likely to occur with rapid bolus administration.

Bretylium tosylate is indicated for ventricular tachycardias (VT) and VF that are unresponsive to defibrillation, epinephrine, and lidocaine. Randomized studies comparing the efficacies of bretylium and lidocaine have failed to demonstrate superiority of bretylium in any significant respect.[12] It is the drug of last resort where VF is refractory or recurrent, and its administration is to be followed within 30 to 60 seconds by application of direct current countershock.

Beta-adrenergic blocking drugs reduce the incidence of VF in post-MI patients. Atenolol, metoprolol, and propranolol are recommended in the guidelines. Following thrombolytic therapy, they may reduce the occurrence of MI, but their routine use is not advised. Bradyarrhythmias, hypotension, and congestive heart failure are contraindications. Also, beta blockers should be withheld from patients with hyperreactive airways predisposing to bronchospasm. The incidence of postinfarction ventricular arrhythmias may also be reduced by infusions of magnesium sulfate, but the presence of hypomagnesemia should first be demonstrated.[13]

Atropine retains its Class I status for treatment of symptomatic sinus bradycardia, and may be of benefit for nodal atrioventricular (AV) block (Class IIa)[14] or ventricular asystole.[15] Isoproterenol, a beta-adrenergic agonist once widely used as a cardioaccelerator, is now recommended only for temporary control of bradycardia in denervated hearts of transplant patients and for refractory torsade de pointes (Class IIa).

Verapamil and diltiazem, calcium-channel blocking agents, retard conduction and increase refractoriness of the AV node. They are indicated for treatment of re-entrant arrhythmias that include AV nodal conduction. Given for arrhythmias of supraventricular origin, they may control ventricular response rates. However, they can decrease myocardial contractility and may worsen congestive heart failure. The arrhythmia to be treated should be identified with certainty as supraventricular. Deaths have been reported following administration of verapamil to patients with Wolff-Parkinson-White

syndrome and with wide-complex tachycardias.[16] Verapamil is given to patients with narrow-complex PSVT but is second choice to adenosine.

Adenosine was unavailable in the United States until 1990. It is an endogenous compound that is rapidly metabolized, with a half-life less than 10 seconds. It depresses nodal activity, can terminate arrhythmias involving the AV node,[17] and is as effective as verapamil in the initial conversion of PSVT.[18] Because its duration of action is extremely short, it produces only minor hemodynamic changes. No untoward effects have been reported, even when patients were given adenosine and the arrhythmia was subsequently identified as VT.[19] If the PSVT is not terminated, repeated doses may be given, or a calcium-channel blocker may be used. Patients taking theophylline may require larger doses of adenosine.[20]

Inotropic Drugs

The naturally occurring catecholamines (epinephrine, norepinephrine, dopamine) and their synthetic congener, dobutamine, are the chief agents used in ACLS to improve cardiac output. Epinephrine (1 mg I.V.) is the first drug to be given in cardiac arrest, following airway management and defibrillation. If this is ineffective, a higher dose (5 mg or 0.1 mg/kg) is a Class IIb recommendation. The interval between administrations, formerly 5 minutes, is now shortened to 3 to 5 minutes. Epinephrine can be given effectively by endotracheal tube in doses 2 to 2.5 times the I.V. dose. Intracardiac administration should be reserved for open cardiac massage, or when other routes of administration are unavailable.[9]

Epinephrine is mainly beneficial because of its alpha-adrenergic receptor stimulating effect. The same is true of norepinephrine, which is indicated for treatment of severe hypotension (< 70 mm Hg systolic) due to low peripheral vascular resistance. The pressor action of norepinephrine is accompanied by marked renal and mesenteric vasoconstriction, with possible resulting ischemia. Dopamine is more versatile, exhibiting both alpha- and beta-agonist activity and specific effects on dopaminergic receptors that may improve renal and mesenteric blood flow. During the period of resuscitation, dopamine is recommended for hypotension caused by bradycardia or after return of spontaneous circulation. Dopamine is less effective than epinephrine in improving hemodynamics during CPR.[21] Recurrent VF preceded by tachycardia may be caused by excessive catecholamine levels. Under these circumstances, the use of a beta blocker may be advisable.

Other inotropic agents include amrinone, calcium salts, and digoxin. The hemodynamic effects of amrinone are similar to those of dobutamine,[22] but it may increase myocardial ischemia and ventricular ectopy. Calcium has limited indications: in hyperkalemic or hypocalcemic states, or when calcium-channel-blocker overdosage has occurred. Digoxin is seldom indicated as an emergency inotropic drug.[23] It can decrease ventricular responses to supraventricular arrhythmias but has largely been replaced by verapamil and diltiazem.[24]

Vasodilators

Two ultrashort direct-acting drugs are recommended, nitroglycerin and sodium nitroprusside. Both must be given by drip infusion in ACLS. The principal indication for their use is to relieve afterload in congestive heart failure secondary to MI. Nitroglycerin may be preferred over sodium nitroprusside in patients with coronary artery disease, because nitroprusside may exacerbate ischemia.[25]

Other Drugs

No comments are offered in this review concerning the uses of furosemide (a potent loop diuretic), thrombolytic agents (four are now available in the United States), or morphine sulfate, which continues to be the drug of first choice for anginal pain and may be given for symptomatic relief in acute pulmonary edema (Class IIb).[26] Recommendations for these agents are essentially unchanged in the 1992 Guidelines.

Sodium bicarbonate now has few indications. The maintenance of alveolar ventilation is the most important factor in the control of acid-base balance during and following the resuscitative effort.[27] There are few data to support the use of alkaline buffers. Defibrillation is not facilitated,[28] hyperosmolarity and hypernatremia are increased,[29] paradoxic intracellular acidosis may result from production of carbon dioxide (which diffuses into ischemic myocardial cells and depresses function), and adverse effects may accompany extracellular alkalosis.[30] For patients with pre-existing metabolic acidosis, hyperkalemia, or overdose with acidic drugs, bicarbonate is definitely beneficial. After extended periods of cardiac arrest, bicarbonate may be helpful (Class IIb); but even when acidosis is severe, complete correction of the base deficit should be avoided.

Pediatric Advanced Life Support

Circulatory arrest in small children presents a spectrum of clinical problems somewhat different from those encountered in adults. Hypovolemia is common, and refractory cardiac arrest may justify administration of a fluid bolus. Bradyarrhythmias are much more frequent in children. At age 6 months or less, cardiac output is rate dependent, which requires that the heart rate be maintained within normal limits. Epinephrine is most often the cardioaccelerator of choice, but atropine may be indicated if the bradycardia is vagally mediated. Hypoglycemia is often present in small children under stress, but glucose solutions should be titrated according to measured

blood concentrations. The restrictions on the uses of sodium bicarbonate in adults apply equally in pediatric life support.

Summary

The 1992 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care are comprehensive. Not only are the minutiae of resuscitation addressed and well documented, but discussions are extended to issues that might be termed sociological. Lifestyle changes are suggested that would tend to decrease the need for life support efforts. Preservation of vital organ function in the post-resuscitation period is considered in detail, and ways to optimize outcomes beyond the critical episode are suggested. The necessity for standardized reporting is emphasized, in order that CPR procedures can more easily be refined and the future necessity for these procedures be reduced.

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Portable Oxygen Therapy: Including Oxygen Conserving Methodology, by Brian L Tiep MD, with 11 contributors. Hardcover, 490 pages, illustrated. Mt Kisco NY: Futura Publishing Co Inc, 1991. $78.00.

In the Foreword of **Portable Oxygen Therapy**, Dr Thomas Petty refers to this work as a source that brings us up to date with all aspects of oxygen therapy. I could not agree more with Dr Petty. The author has given us a comprehensive and easy-to-read book covering both the rationale for and practice of delivering oxygen therapy.

The text begins with a look at the history of oxygen therapy and concludes with a look at future directions of the practice. Between these two sections are five major areas of concentration. Pulmonary Rehabilitation is discussed first with emphasis on its importance as an extension of portable oxygen therapy; goals, screening, and the available modalities are discussed in detail.

Chapters 4 and 5 (on physiologic principles of oxygen delivery and acid-base status, by contributor Rick Carter PhD) are especially well done. Complete with many graphs and tables, these chapters are informative to everyone from student to veteran caregiver.

The chapter on oxygen systems and hazards gives the reader a detailed look at the many available products in the field and lists the limitations and hazards of each. The importance of matching the modality to the patient's lifestyle is stressed.

One of the most comprehensive chapters in the text pertains to transtracheal oxygen therapy. The author gives us a look at an ideal candidate and covers the contraindications for transtracheal delivery. The available systems are discussed in much more detail than in the manufacturers' literature on which many healthcare professionals rely.

The section entitled "Prescribing Home Oxygen" takes the reader through all of the steps in prescribing home oxygen. The information on Medicare regulations, home medical supplies, and oxygen prescriptions should assist the physician in justifying the need for oxygen therapy and ensuring that the home medical supplier is able to carry out these orders.

Oxygen-conservation devices and the electronic demand-delivery devices are foreign to many in the field of respiratory care. The use of these products may increase as reimbursement changes. A large section of this book is dedicated to the theory and application of these devices.

In summary, **Portable Oxygen Therapy** contains something for everyone involved in the implementation of oxygen therapy. The text is well indexed, comprehensive, and readable. This work would be an asset to any respiratory therapy department and an excellent source to use to set up a pulmonary rehabilitation division.

Edward R Hoisington RRT
Shift Supervisor
Department of Pulmonary Medicine
The Cleveland Clinic Foundation
Cleveland, Ohio

Cardiopulmonary Anatomy & Physiology: Essentials for Respiratory Care, 2nd edition, by Terry Des Jardins MEd RRT. Soft-cover, 454 pages, illustrated. Albany NY: Delmar Publishers Inc, 1993. $25.95.

One of the most challenging tasks faced by respiratory care educators is teaching their students the concepts of cardiopulmonary anatomy and physiology that serve as the foundation for understanding respiratory care. With 20 years experience in teaching such a course, Des Jardins has written the second edition of **Cardiopulmonary Anatomy & Physiology: Essentials for Respiratory Care** as an easy-to-read, comprehensive text that effectively addresses this difficult task.

In the Preface, Des Jardins indicates that the book is written from the premise that the reader has no previous knowledge of respiratory anatomy and physiology, and the structure of the text honors this claim. Each chapter is preceded by a detailed set of objectives designed to assist the reader in identifying the important concepts to be addressed. Within each chapter, key words appear in bold print and are defined. The glossary is comprehensive and serves as a quick reference for definitions of medical terminology. The abundance of illustrations and tables clarify and provide visual representation of anatomical structures, key concepts, and normal values (46 illustrations and tables appear in Chapter 1 alone). Furthermore, self-assessment questions are provided for each chapter, enabling readers to quickly and painlessly check their understanding of the material. Answers to these questions are provided in the appendix. (The publisher advised me that a student workbook, $13.95, and instructor's manual, $16.00, are to be available in the near future.)

This text has excellent breadth and depth of subject matter, and the author has utilized a bibliography of 142 entries to validate the text's contents. The bibliography is organized by chapter and could serve as a reference for supplementary reading.

Chapter 1 provides a presentation of respiratory anatomy that rivals other college-level anatomy text-

books. Chapters 2 through 10 provide the crucial elements of respiratory physiology. Chapter 2 provides a discussion of ventilation that includes static and dynamic characteristics of the lungs and ventilatory patterns. Chapter 3 explains the diffusion of pulmonary gases and is followed by an overview of pulmonary function measurements, in Chapter 4. Chapter 5 presents the seminal concepts of the circulatory system and precedes the discussion of hemodynamic measurements provided in Chapter 6. Chapters 7 and 8 discuss oxygen and carbon dioxide transport and acid-base balance and include clinical case studies to facilitate learning and application of the concepts. Chapter 9 presents a discussion of ventilation-perfusion relationships, and Chapter 10 addresses the control of ventilation.

Chapter 11 provides an excellent overview of the cardiopulmonary physiology of the fetus and the newborn that could supplement an advanced neonatology/pediatrics respiratory care course. Frequently troublesome topics for students—renal physiology and renal failure and its effects on the cardiopulmonary system—are presented in a concise and easy-to-understand manner in Chapter 12. Chapter 13 completes the life cycle discussion of respiratory anatomy and physiology, presenting the effects of aging on the cardiopulmonary system.

Chapters 14, 15, and 16 are new chapters in this edition. Chapter 14 provides a discussion of exercise and its effects on the cardiopulmonary system and could serve to introduce the study of exercise testing. An explanation of the effects of high altitude on the cardiopulmonary system is found in Chapter 15. The text concludes with an overview of high-pressure environments (Chapter 16), including decompression sickness and hyperbaric medicine and their effects on the cardiopulmonary system.

In comparison to the first edition, several differences are notable. First, the second edition is approximately 150 pages longer, the three new chapters account for 38 pages. The remaining increase in length can be attributed to additional and improved spacing of illustrations and tables. In most cases, only one illustration appears on each page (Page 89, for example) in contrast to the presentation of multiple illustrations per page in the first edition. Some of the illustrations have been enlarged (Page 95); others have been redrawn utilizing less contrast/shading (Pages 14 and 22). The overall effect provides more white space and, I believe, decreases reader confusion regarding which illustration or table corresponds with the text. In addition, a page number is provided for illustrations and tables that do not appear adjacent to the corresponding text (Figure 2-26, Page 96), making it easier for readers to locate the desired information.

A second difference is a change in the sequencing of chapters. A discussion of the diffusion of pulmonary gases (Chapter 3) now precedes the chapter on pulmonary function measurements (Chapter 4). This move is appropriate because Chapter 4 now includes a discussion of the diffusion capacity of carbon monoxide (D_{LCO}), and a prior explanation of the diffusion of pulmonary gases facilitates understanding of this measurement.

Additional improvements include the provision of more comprehensive objectives for Chapters 1, 2, and 3; the addition of self-assessment questions for Chapter 6; and the presentation of the self-assessment questions in a single column rather than the double-column format utilized in the first edition.

A minor but inexcusable weakness in the text is the inaccuracy of the table of contents. The page numbers are in error by as much as six pages (with an average discrepancy of two or three pages) for each content area. As an example, a topic entitled "Overview of Specific Ventilatory Patterns" in Chapter 2 is listed as beginning on Page 111 but actually begins on Page 105. This could be frustrating to readers as they attempt to locate a particular topic. However, in contrast, the index provides accurate information and page numbers and therefore could be very helpful as a quick reference for locating the desired information.

In summary, Des Jardins successfully accomplishes his goal in writing this text. **Cardiopulmonary Anatomy & Physiology: Essentials for Respiratory Care** provides an excellent, easy-to-read, comprehensive presentation of subject matter essential to understanding respiratory care theory and practice. It is suitable for respiratory care students and practitioners, medical students, and other allied health students or professionals who desire an in-depth understanding of this topic.

Cynthia A Beckett MS RPFT RRT
Professor and Chairperson
Respiratory Care Program
Sinclair Community College
Dayton, Ohio

Respiratory Disease: Principles of Patient Care, edited by Robert L Wilkins MA RRT and James R Dexter MD. Hardcover, 414 pages, illustrated. Philadelphia: FA Davis Co, 1993. $35.00.

Somehow the odor, the texture, the visual impact of a new book touches the soul of a bibliophile like no other stimuli. These intangibles are measurably heightened when the content is right. And, to this reviewer, **Respiratory Disease: Principles of Patient Care** is right!

In my opinion, Wilkins and Dexter (both associated with Loma Linda University in California) and their 10 contributors have produced a singularly unique text for advanced respiratory care students. This textbook should also have a broad appeal for medical and nursing students inter-

ested in the broad range of pulmonary disorders, as well as clinical practitioners seeking to improve their patient assessment and management skills. Thomas A Barnes EdD RRT (of Northeastern University in Boston) ended his Foreword to this book by writing, "I plan to make this a required text for a course I teach called 'Respiratory Care for Medical and Surgical Patients.'"

For the pure joy of it, I approached my critique of this volume as any student would. Armed with a note pad and a supply of colored highlighters, I turned to the Table of Contents. It is worth listing the 23 Chapters here because it illustrates the scope and thrust of **Respiratory Disease**: Introduction to Respiratory Failure, Asthma, Chronic Bronchitis, Emphysema, Cystic Fibrosis, Hemodynamic Monitoring and Shock, Pulmonary Thromboembolic Disease, Heart Failure, Smoke Inhalation and Burns, Near Drowning, Adult Respiratory Distress Syndrome, Chest Trauma, Postoperative Atelectasis, Interstitial Lung Disease, Neuromuscular Disorders, Bacterial Pneumonia, Lung Cancer, Sleep Apnea, Croup and Epiglottitis, Respiratory Syncytial Virus, Respiratory Distress Syndrome in the Newborn, Bronchopulmonary Dysplasia, and Persistent Pulmonary Hypertension of the Newborn. Valuable features, especially for students, are the Glossary and the Tables of Normal Laboratory Values.

Each chapter is constructed on a framework of introduction, etiology, pathophysiology (and pathology where necessary), clinical features, diagnosis, treatment, case study, and references. The case studies are expanded to include relevant questions about important features of the cases followed by a section offering the authors' answers to the questions posed. In this way, the reader is compelled to think about the issues presented and to structure an answer. By comparing his answers to those in the text, the reader learns and reinforces the material. By the time I finished Chapter 3 (Chronic Bronchitis), I was somewhat surprised to realize how close my answers were becoming to those of the authors.

The syntax is pleasurably readable. It is remarkably free of clinical officiousness and convoluted explanations. The authors have used the simple declarative sentence with a degree of elegance to explain and to teach. The chapter headings and subheadings are boldly defined, a definite aid to the reader—particularly one with aging eyes like mine.

My current professional focus is on sleep disorders. I studied Chapter 18, Sleep Apnea, with particular zeal. I was disturbed to note that the authors of this chapter failed to acknowledge the manual by Rechtschaffen and Kales (R & K),[1] the 'gold standard' for sleep staging and scoring, in their discussion of sleep and breathing. Perhaps this failure accounts for a troubling Figure 18-3, which purports to illustrate the onset of Stage REM (rapid eye movement) sleep. This figure does show unambiguous Stage REM; however, according to R & K, "Sleep spindles and K complexes stop and the EEG changes to relatively low voltage, mixed frequency for one or more epochs before REMs start. Score all the record from the last sleep spindle or K complex as Stage REM . . .."[1]

The value of a glossary should not be underestimated, even to advanced students. I feel that this one should be expanded to include such terms as JVD, jugular venous distention (Page 34) and NIPPV, nocturnal nasal-mask intermittent positive-pressure ventilation (sic) on Page 257, as examples.

The line drawings and illustrations throughout the book are uniformly excellent with one exception. In Figure 1-2, the cartilaginous rings surrounding the trachea are highly stylized. This suggests a construct that is simply erroneous in human anatomy.

The major fault—if a fault it truly is—in this textbook is the paper upon which it is printed. It is very thin and porous. My highlighting fluid, mainly the color pink, soaked through the pages to the succeeding ones. Too, because the print "bleeds" through the pages, some of the illustrations are distorted. A case in point is Figure 14-5 (Page 237), a radiograph showing "honeycomb lung." In my copy, a peculiar anomaly over the heart shadow turned out to be the bold "Prognosis" subheading bleeding through from Page 238.

On the other hand, as a beleaguered parent with a son in college, I well appreciate the effort made to keep the cost of this book at a minimum: $35.00 is a very attractive price for this text.

In summary, the editors of **Respiratory Disease: Principles of Patient Care** open their Preface thusly, "This text is written to help the reader understand the assessment and treatment of patients with respiratory disease." They have succeeded. I am pleased to have this volume in my library. I look forward to future editions with anticipation, and would be happy to pay a few dollars more for improved paper quality.

Robert R Weilacher RRT
Technical Manager
East Texas Sleep Center
Medical Physiology Department
Memorial Hospital
Palestine, Texas

REFERENCE

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The Quality of Compressed Air for Sprays and Inhalations

(A Report in JAMA 100 Years Ago)

Asepsis is now in the zenith of its usefulness and has demonstrated beyond controversy its beneficence to all unfortunates who have to experience the surgeon's dextrous touch or feel his keener blade; every newly made wound is carefully guarded against the ingress of air, of any kind, and quickly sealed against it; the surgeon, gynecologist, or accoucheur goes about his work now, with a comfortable assurance that the painstaking physician must envy in conscious helplessness when he recognizes the environment in which he must combat disease.

There is an avenue, however, which for the introduction of disease and death growing germs has no parallel and no excuse for its existence, other than that it is here, and no one has protested against it.

The allusion made, is in regard to the quality of air usually used in the compressed air receivers of those who treat diseases of the nose, throat, and lungs by this means; all the air pumps I have observed offered for sale or in physicians' offices—and I have taken special pains to see as many of them as possible—are situated on the floor of the doctor's office, in not an overclean corner from which the air is drawn into the pump and thence stored in the reservoir to be afterwards projected with a pressure of from fifteen to twenty pounds into the diseased larynx or pharynx of the innocent patron whose inflamed crypts are deep and open wells for the speedy reception, and successful propagation of any of the germ diseases; with just the right factors of moisture and heat to develop them all; or, if drawn into the deeper structure of the lungs to mingle with, or become a portion of the residuary air, a sufficient period would ensue before again exhaled, for the successful incubation of almost any germ, and a nidus for dangerous disease be formed.

To ease my conscience of this offense against my patients I had a mechanic of the Air Brush Co., of this city, at my suggestion, construct for me a small oscillating pump which is run by a one-eight horse power electric motor, that draws the air from out of doors; first through a five inch funnel-shaped chamber, whose open end is covered with wire gauze and its inside loosely packed with asbestos wool to make a complete filter; this filter chamber is fastened at the outside top of a window sash of an upstairs office, and is connected by a rubber tube, larger than would seem necessary—to reduce resistance to a minimum—to the inlet valve of the pump, and is thence, at the return of the piston, forced into the air receiver for future use; by this means all the air comes from outside, is filtered, and it is claimed, that cleaner air is obtained than by the pumps generally located on doctors' dusty office floors.

The mechanical difficulties necessary to adjust any air pump to a similar filter system are not great, or expensive; and it is hoped that the physician will be enabled to use as aseptic air to inhale, or to spray diseased throats as the surgeon does to douche his patients' wounds.

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*The material on which this report is based originally appeared in JAMA, Jan 28 (1893;20:89) and was written by D Lichty MD of Rockford, Illinois. The version presented here is reprinted, with permission, from "JAMA 100 Years Ago," in JAMA—the Journal of the American Medical Association, Jan 27, 1993; 269:446, having been edited by Annette Flanagin RN MA, Associate Senior Editor of JAMA, and Micaela Sullivan-Fowler MS MA. Copyright 1993, American Medical Association.

Letters

Letters on topics of current interest or commenting on material in RESPIRATORY CARE will be considered for publication. The Editors may accept or decline a letter or edit without changing the author's views. The content of letters as published may simply reflect the author's opinion or interpretation of information—not standard practice or the Journal's recommendation. Authors of criticized material will have the opportunity to reply in print. No anonymous letters can be published. Type letter double-spaced, mark it "For Publication," and mail it to RESPIRATORY CARE Journal, 11030 Ables Lane, Dallas TX 75229-4593.

Objection to Case Report of Nishizawa et al

In the case report "Pulmonary Edema Induced by Endotracheal Tube Obstruction. . .",[1] the authors state that the patient could not be effectively ventilated for approximately 3 minutes. The authors describe a patient who was flailing his extremities, biting furiously on the endotracheal tube, making vigorous inspiratory efforts against the compressed tube, and experiencing a desaturation from 98 to 65%. During this acute emergency, the only intervention attempted by the clinicians was the insertion of an oral airway as a bite block! Finally after 3 minutes had elapsed and an opiate antagonist had been administered, the attending clinicians were able to successfully place an oral airway and ventilate the patient.

Nowhere in the report do the authors mention the most obvious action to be taken: immediate deflation of the cuff and attempted extubation of the patient!

I find it hard to accept that this patient who had apparently recovered enough from his anesthetic agents to have a respiratory drive was left to struggle for 3 minutes against an occluded airway.

Deflating the cuff would probably have provided enough airway patency to prevent the pulmonary edema in the first place and with the cuff deflated the patient might have relaxed his bite enough to allow extubation. In a less stable patient with more cardiovascular compromise, 3 minutes of complete airway obstruction could have resulted in cardiac arrest and a far less favorable outcome for the patient.

I did not personally witness the event and must react solely to the case report as it has been described, but if my suggestion was attempted it should have been mentioned. Otherwise, although this case report has value as a discussion of pulmonary edema induced by an obstructed airway, a valuable lesson in airway management has been missed entirely.

Phil Mercurio RCP RRT
Quality Assurance & Equipment Supervisor
Respiratory Therapy Department
UCLA Medical Center
Los Angeles, California

REFERENCE

1. Nishizawa M, Chaffee TL, Goto H. Pulmonary edema induced by endotracheal tube obstruction: a case report. Respir Care 1993;38:479-481.

Dr Chaffee and Dr Goto respond:

We thank Mr Mercurio for his valuable comments on our case report.[1] Indeed, in the discussion surrounding this case, we entertained all therapeutic options to avoid negative pressure pulmonary edema (NPPE) including deflation of the endotracheal tube cuff.

Our decisions were tempered by the implications of emergence from an inhaled anesthetic, a clinical situation that is different from that of a combative patient on a ventilator. Patients in this stage of anesthesia may appear vigorous but are highly prone to laryngospasm and unable to protect their airways against aspiration. For the anesthesiologist to give up access to such an airway and to leave the airway unprotected by extubation is potentially hazardous.

We agree that deflation of the cuff could possibly have allowed some minimal air passage around the endotracheal tube. However, the space between the vocal cords and the endotracheal tube would have been limited, and airflow would have been turbulent and inadequate with this patient's vigorous inspiratory efforts. Consequently, the patient might still have developed high negative intrathoracic pressures, with the added risk of having the airway unprotected. Management decisions must weigh those risks against a potential minimal benefit.

The focus of our case report was the rapidity with which NPPF can develop and a discussion of its apparent mechanisms. We agree with Mr Mercurio that deflation of the endotracheal tube cuff could have been mentioned as another potentially useful, yet risky, intervention.

Terry L Chaffee MD
Assistant Professor

Hiroshi Goto MD
Professor

Department of Anesthesiology
University of Kansas Medical Center
Kansas City, Kansas

REFERENCE

1. Nishizawa M, Chaffee TL, Goto H. Pulmonary edema induced by endotracheal tube obstruction: a case report. Respir Care 1993;38:479-481.

Complication due to a Central Venous Catheter

A practical problem associated with the central venous catheter and the differential diagnosis of widened mediastinum has been effectively addressed by Woodruff and Clevenger.[1] However, we would like to address some of the points made in their presentation.

The authors mention that serial chest radiographs were found to be normal until the eighth day, but would it not have been appropriate to show the radiograph shown on the seventh day in Figure 1, rather than one selected arbitrarily from the se-

ries? That would have supported the probable acuteness of the problem.

The authors also mention that before removing the leaking central venous catheter that they elevated the head of the bed to 45° to reduce the central venous pressure. But, isn't it standard practice to keep the head of the bed elevated to at least 30° in patients who sustain head injuries (confirmed in this patient by the presence of edema and the appearance of the computed tomography scan)? So, was it necessary to elevate the head of the bed another 15°?

Despite the authors' stressing the importance of immediate diagnosis of fluid extravasation into the chest, no comment was made regarding a simple, practical solution to the problem. Treatment for mediastinal widening was elaborated upon, but it is important to stress that the goal should be to diagnose as early as possible. One cannot always depend on clinical signs because a majority of head-injured patients who require central venous catheter placement will be on mechanical ventilation because of altered sensorium. Instead of waiting to see a mediastinal widening in such patients, a simple, practical solution would be to aspirate the catheter every 6-8 hours to make sure that the content is blood. If fluid other than blood is aspirated, other steps mentioned by Woodruff and Clevenger[1] can be undertaken. This does not mean that a daily radiograph may not be desirable.

Regina Nerida RN
Shankar P Gopinath MD

Department of Neurosurgery
Baylor College of Medicine
Houston, Texas

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Dr Clevenger responds:

Ms Nerida and Dr Gopinath have raised several interesting questions related to the management of the blunt trauma case described in our paper.[1] Although the legend for Figure 1 reads "one of a series of chest radiographs taken during the first week. . .," it would have been correct to specify the timing as they suggested. Indeed, the chest x-ray shown as Figure 1 was taken on the seventh day of the patient's admission (24 hours prior to the chest x-ray in Figure 2, which demonstrates the abnormality). We apologize for any confusion caused by this caption; however, we wanted to make it clear that the patient's X-ray pattern had been stable for the 7 days prior to the onset of the abnormality discussed in the paper.

Elevation of the head of the bed is actually not the standard of care in our unit and based on my personal communication with other surgeons may not be in a number of neurosurgical intensive care units around the country. Although elevation of the head of the bed does decrease intracranial pressure in head-injured patients, it has a variable effect on cerebral perfusion pressure. We work to maximize cerebral perfusion pressure and indeed many patients with closed-head injury are nursed with the head of the bed at 0° (flat). This approach to head injury management was originally advocated by Rosner et al.[1] In our patient's case, elevation of the head of the bed to 45° was required prior to removal of the central line because he was being nursed with the bed flat.

The final concern of Ms Nerida and Dr Gopinath is over the failure to routinely aspirate the catheter every 6 to 8 hours to make sure that the content is blood. Unfortunately, an intravascular catheter perforation may be associated with a large pseudoaneurysm, leading to the accumulation of enough blood around the tip of the catheter to allow aspiration of blood despite vessel perforation. In addition, even in the absence of a pseudoaneurysm, regular breaking of the line is believed by some to dispose patients to the development of sepsis. Breaking into a catheter system (particularly a line for total parenteral nutrition, or TPN) has been shown to increase the incidence of infectious complications.[2,3] The suggestion of Ms Nerida and Dr Gopinath to physically break the central line system 3 or 4 times per day would predispose to infectious complications, and we believe that it should not be advocated as a matter of routine.

We appreciate the interest in our article and the suggestions put forth by Ms Nerida and Dr Gopinath. Further, we appreciate the opportunity to respond to their comments.

Frederick W Clevenger MD
Associate Professor of Surgery
Chief, Burns & Trauma Division
University of New Mexico
Albuquerque, New Mexico

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Rewarming with Gas Mixtures?

I was very interested in the editorial by Chatburn and Branson concerning humidification while on me-

chanical ventilation.[1] The part that interested me most was the section entitled, "Can Adjusting Inspired Gas Temperature Affect Body Temperature?" I manage a respiratory care department in rural Alaska, and Kodiak is a seaside town whose main business is commercial fishing. The waters around Kodiak are cold year-round, and we see more than our fair share of cold-water near-drowning and, to a lesser extent, hypothermia. I followed the authors' line of reasoning and agree with their basic assertion.

I believe that the problem now is How do we as a profession convince the physicians and Emergency Medical Technicians that they no longer should view the administration of heated and humidified oxygen as viable rewarming technique? I just attended a lecture in Kodiak where I asked Dr Martin Nemiroff, a noted expert on cold-water near-drowning, how he felt about heated oxygen as a rewarming technique. He said that he was aware of the argument that the respiratory care community was voicing but, "I am more interested in cor rewarming than core rewarming."

I believe the basic problem to be that heated oxygen has become the standard of care for hypothermic patients, and clinicians deviate from accepted standards at their own peril. The fact is that most people view heated oxygen for rewarming as a safe effective treatment.

I encourage the authors of that editorial as they take an important first step in debunking a popularly held misconception. I also hope that consensus statements and Clinical Practice Guidelines for hypothermia and cold-water near-drowning will be developed in the near future. I recommend that we start talking to our physician colleagues about this issue as soon as possible. I feel that physicians as a whole are unaware of the data and arguments presented in the editorial. I as a clinician and manager would like to stop wasting time and resources on an intervention that cannot work and may be harmful.

Wayne Wallace BA RRT
Respiratory Care Manager
Kodiak Island Hospital and
Care Center
Kodiak, Alaska

REFERENCE

1. Branson RD, Chatburn RL. Humidification of inspired gases during mechanical ventilation (editorial). Respir Care 1993; 38(5):461-468.

Dr Nemiroff responds:

The use of humidified, heated gas should not be considered a total body rewarming technique as such. I view it more as a stabilization technique, warming the heart 1 or 2° C relative to the body temperature. I believe that this has the salutary effect of reducing the excitability of the heart, reducing ectopy, and reversing the bradycardia one sees with hypothermia. Overall, it is an effective technique in the treatment of hypothermia but one should not mislabel it as a body rewarming technique. Caution should be observed and cardiac monitoring instituted as rewarming is undertaken. Rewarming dysrhythmias are common as peripheral stores of cold, acidotic, metabolite-rich blood reenter the circulation during the rewarming process.

CAPT Martin J Nemiroff MD
Commander, MLC Pacific
CG Island
Alameda, California

A Stylet To Aid Digital Intubation

I enjoyed the excellent article by Dr Kovac in the April issue of the Journal.[1] I was especially interested in the section on digital, or tactile, orotracheal intubation. The method so clearly described by Dr Kovac, which uses only the fingers and the endotracheal tube (ETT), is excellent for those with long fingers but is somewhat difficult for those with short fingers. Sometime ago, I (because I have short fingers) designed a curved, springy stylet to aid digital orotracheal intubation.

The technique used with the stylet is the same as that presented in Dr Kovac's article except that the operator first inserts the anteriorly curved stylet digitally through the glottis and then slides the preloaded ETT over the stylet into the trachea. The stylet is then removed. We have found that the stylet makes it reasonably easy for those with short fingers—or with long ones—to intubate successfully and have presented our findings in abstract form at poster sessions.[2,3] The device can be obtained from Cook Co Inc, 925 S Curry Pike, Bloomington IN 47402.

Pasquale Ciaglia MD
General Thoracic Surgery
& Endoscopy
Utica, New York

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Humidification and Colds: My n = 1

About 5 years ago, the idea came to me that cold viruses might enter

the nasal mucosa solely because of inadequate humidification. I decided to conduct a simple experiment using myself as subject (my n = 1).

In August 1988, I began to use over-the-counter saline nasal spray (0.65% solution, any brand) in each nostril 6-8 times each day, throughout the year. In the ensuing 5 years, I have contracted only 2 colds. Although this may not be statistically significant, it is important to me (and may be to others also) because since childhood (I am now 43 years of age), I have always contracted 2 colds a year, one in late Fall and another in early Spring.

In all probability, my minimal rate of infection is nothing more than good luck, but I decided to report my experience via letter, hoping that someone in the scientific community might further investigate the subject with an adequate sample and controls.

Raymond F Groff BA RPFT RRT
Columbia, Pennsylvania

Misrepresentations in the Marketing of Heat and Moisture Exchangers Continue

In 1981 in letters published in RESPIRATORY CARE,[1,2] I called the attention of respiratory clinicians to what I believed to be misleading and false claims for the performance of the Siemens Servo HME. Subsequently, many therapists expressed their appreciation to me for presenting that information.

For the last year or so, I have been concerned about statements that appear in a Technical Report prepared and distributed by the Pall Corporation in support of their product.[3] I believe that Report misrepresents the clinical value of the Pall HME and incorrectly defines the role of heated humidifiers in nosocomial infections.

The mistakes of the past are being repeated.

I believe that the Report incorrectly describes how a Cascade humidifier operates, incorrectly describes a heated humidifier as providing an environment favorable for the growth of a variety of pathogens, incorrectly implicates circuit tubing as a means to transmit organisms from heated humidifiers, and incorrectly claims that the Pall HME is an effective alternative to heated humidifiers for most patients. It further incorrectly states that only a small number of long-term ventilator patients require humidity levels higher than an HME alone can deliver and claims that a cold humidifier used with an HME can supply adequate humidity "to this special group of patients." The Report includes data collected from experimentation with a patient simulator that, I believe, overstates the performance of the Pall HME.

The possible airborne movement of pathogens from ventilator-dependent patients to the environment is an important clinical issue, and according to the Technical Report the Pall HME prevents such movement. However, the source of those pathogens is not the reservoir of the heated humidifier as the Technical Report states but rather the patient. The notion that the reservoir may be the source may stem from a misinterpretation of the results from a paper by Dyer and Peterson[4] reporting that positive pathogen cultures were obtained on plates as far as 32 feet from an IPPB machine. That paper "How Far Do Bacteria Travel from the Exhalation Valve of IPPB Equipment?" clearly explains that the system tested incorporated a medications nebulizer. Heated humidifiers are not particle generators but nebulizers are.[5,6] Further, use of the Pall HME as an 'add-on' expiratory filter may be redundant and unwarranted because the Puritan-Bennett 7200 ventilator series incorporate a heated expiratory filter before the exhalation valve, and other ventilators have been equipped with external expiratory filtration devices, such as the Infrasonics STAR system, which provide the same filtration benefit.

The Pall Technical Report states: "Cascade humidifiers operate by bubbling inspiratory air [sic] through water in the chamber, generating an aerosol of water droplets". Without additional comment about the misnomer (ie, use of the term air when gas is correct), the statement demonstrates a lack of understanding of the operation of a formerly most ubiquitous piece of respiratory equipment. Most of the moisture added to inspiratory gas by a Cascade-type humidifier is the result of evaporation of water from the entire heated-water surface. The Cascade grid is used to increase water surface area for contact with the incoming gas by causing the gas to bubble through a thin layer of water. The Cascade concept allows the humidifier to heat and saturate incrementally higher gas flows. Without the grid, the gas does not bubble through the water, and the Cascade humidifier functions as a pass-over humidifier. Many of us probably have had personal experience with the successful ventilation of patients with the Cascade tower removed.

Condensate in ventilator tubing becomes contaminated from pathogenic material ejected by the patient. Pathogens may be introduced into a previously sterile heated humidifier by the introduction of contaminated condensate from the inspiratory tube. However, if a humidifier does become contaminated, the literature shows that this is a benign event.[7-12] Studies on the role of respiratory equipment in nosocomial infections have revealed the inability of pathogens to migrate from heated humidifiers, especially wick-type units; and that humidifier water operating temperatures do not support pathogen survival. Using data from these stud-

ies,[7-12] Craven, who to my knowledge has reported most often on this subject, has unequivocally stated[13] there is no colonization in humidifiers whether they are wick-type or bubbling. Comhaire and Lamy[11] reported no growth in all heated humidifiers used in their study of 25 patients, even after 20 days of continuous use. The manufacturer and model number of the heated humidifiers were not disclosed, other than a mention of the Engstrom ventilator and its humidifier. No other special procedure, such as heating at 60°C, was performed. The Pall Report ignores those studies in claiming that condensate forming in circuit tubing can transmit pathogens from the water in a heated humidifier.

The Pall Report misrepresents the performance of the Pall HME, in the claim that "it is an effective alternative to heated humidification for most patients." The Pall Report overstates the Suzukawa et al study[14] in that Suzukawa et al limit their recommendation for the use of the Pall HME and cold humidifier to "appropriately selected long-term ventilator patients in the ICU." At least two clinical papers present evidence that Pall HMEs, without a cold humidifier, are not suitable for most patients.[15,16]

Cohen et al[15] reported 15 episodes of tracheal tube occlusion in 15 patients of a group of 170 patients ventilated with the Pall HME but only 1 occlusion in the group of 81 patients ventilated with Bennett Cascade heated humidifiers. The incidence of occlusions with the Pall HME was therefore 7 times higher (odds ratio = 7.70). They also reported that the Pall HME was frequently replaced with a heated humidifier because of inadequate humidification. They concluded that the Pall HME does not provide adequate airway humidification for general ICU use and that its role outside the operating room remains to be determined.

Martin et al's study[16] was a prospective, randomized, controlled study comparing the Pall HME to heated humidifiers. They reported 6 episodes of tracheal-tube occlusion in 6 patients in the HME-ventilated group of 31 patients. No occlusion occurred in the group of 42 heated-humidifier-ventilated patients (odds ratio = 10.1).

Two additional important facts are disclosed in the Martin et al study.[16] The study was interrupted after the death of one patient in the Pall HME group from total obstruction of the tracheal tube. Also, the number of days of hypothermia in the HME group was almost twice as high (23%) as in the heated-humidifier group (12%). Although the role of gas temperature in body temperature control is controversial,[17] because the heated humidifiers were set to deliver inspiratory gas at 31±1°C, the hypothermia observed in the heated-humidifier group might have been ameliorated by increasing the inspiratory gas temperature. Of course, no compensation is possible with an HME. Interestingly, I do not recall seeing a mention, let alone a discussion, of hypothermia in any HME manufacturer's literature.

A comment in the Cohen et al study,[15] fully contradicts the claim made by Pall for the universal utility of their HME with all mechanically ventilated patients. Cohen et al observed: "ICU patients are a heterogeneous group. Those requiring mechanical ventilation are often unstable and have higher metabolic rates. A large proportion of patients on mechanical ventilation will require high respiratory minute volumes during the course of their illness and may therefore represent a group predisposed to tracheal tube occlusion with HMEs." They concluded: "Based on our experience with the PUBF [Pall Ultipor Bacteria Filter], we conclude: (a) these devices are not adequate for the universal replacement of heated water humidification systems; (b) this is especially true in patients requiring delivered minute volumes >10 L/min, $F_{IO_2} > 0.4$; and (c) if used, they should be replaced by conventional humidification when difficulty in suctioning is encountered". Martin et al's conclusion[16] also cited inadequate humidification of inspired gas by the Pall HMEs.

I believe that the Pall Report's claim for the near-universal utility of their HME is contradicted by clinical reality. Branson and Hurst,[18] in their evaluation of 7 HMEs, found the Pall Conserve HME to be only the third or fourth most efficient in moisture exchange at flowrates of 0.5, 0.75, and 1.0 L/s, and F_{IO_2}s of 0.21 and 1.0. In addition to expressing concern about increased resistance when the hygroscopic insert absorbs water, they found that HMEs cause a net water loss from the patient, which they observed as undesirable in many patients.

Many patients requiring mechanical ventilation present with retained secretions. Their respiratory systems cannot supply adequate humidity, and they may be dehydrated. Cohen et al[15] reported that frequent, unrecorded changeovers from HMEs to heated humidifiers occurred, that the real risk of tracheal tube occlusion is almost certainly much higher, and that there was a detectable increase in atelectasis, pneumonia, and duration of mechanical ventilation associated with HME use. The Pall system may meet the minimum level of acceptable moisture output; however the output of the system cannot be increased. Many patients will require higher levels of inspiratory humidity. If the system is used on those patients, it will not meet their needs, and a physiologic insult will be imposed upon the patients.

A study by Primiano et al[19] concludes that the Siemens Servo HCH and the tracheal tube produced in-

spiratory gas temperatures and moisture contents, at the carina, less than nose-breathing but higher than mouth-breathing. In commenting on this conclusion, Realey[20] questioned the clinical significance of this conclusion in the light of his experience described in his co-authored report of a bronchial cast ejected by a patient, in the course of several violent expiratory maneuvers, on the eighth day of ventilation with a Servo HCH.[21] This paper also reported a marked increase in aspirated secretions after the HME was replaced with a heated humidifier.

Finally, the Pall Technical Report discloses that the patient simulator used to determine water loss measurements, when the Pall HME was tested alone and with 3 different cold humidifiers, was set to deliver expiratory gas to the HME at 33-38°C—a range that simply is not seen clinically. The Primiano et al paper[19] reported an average end-inspiratory temperature of 27.4 ±1°C and an average end-expiratory temperature of 32.5 ±1.5°C for the 5 patients in their study. Branson and Hurst,[18] citing prior studies by Ingelstedt[22] and Dèry et al,[23] used an 'expired' gas temperature of 32 ± 1°C. If laboratory data are to be used to project the clinical performance of a device, the laboratory simulation should replicate in-vivo conditions. It is obvious that the temperature setting of the patient simulator greatly overstates in-vivo conditions; skews the data so as to overstate the actual performance that may be expected in vivo; and renders the Pall Report data meaningless.

As flawed as I believe the Pall Report to be, it does prove my contention that all HMEs, whether used with a cold humidifier or alone, impose a moisture demand upon the patient. As stated earlier, Branson and Hurst[18] report this as fact. The Pall Report data show a water loss from the "artificial patient" when the HME was used alone or with a cold humidifier. This is how secretions become inspissated when HMEs are used. The Pall Report says nothing about replacing the HME with a heated humidifier when secretions thicken, but Realey[20] has expressed the most cogent response to that recommendation I have yet seen. "Why is this procedure necessary at all? Should the patient be required to demonstrate increasingly thick, tenacious secretions prior to being provided with adequate humidification of the inspired gas? We think not." Realey concludes: "What should result from this dialogue is an understanding that the basic principles of high-quality patient care must be of the utmost importance to all of us practicing respiratory care. These principles are applied most clearly, in this case, in the need to provide adequate humidity to the patient's airway during mechanical ventilation."

Bernard R Paluch
President
Nova-VentRx Inc
Northbrook, Illinois

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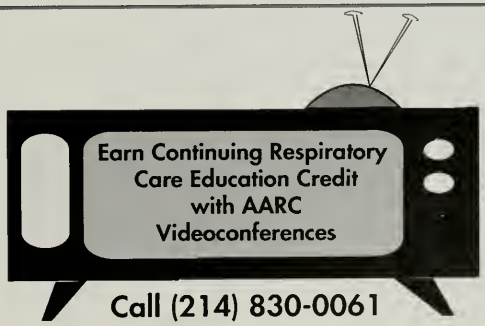

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For the AARC Annual Meeting Nashville, Tennessee

December 11-14

OREGON

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The Oregon opportunity combines state-of-the-art medicine with unlimited outdoor recreational opportunities. Join us in beautiful southern Oregon and experience the challenge!

Rogue Valley Medical Center is a progressive 305-bed non-profit regional trauma center serving 9 counties of southern Oregon and northern California.

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Full-time position for RRT with NBRC, NRP and PALS certification. Oregon license/eligibility. Neonatal transport experience and perinatal/pediatric specialist credentials preferred.

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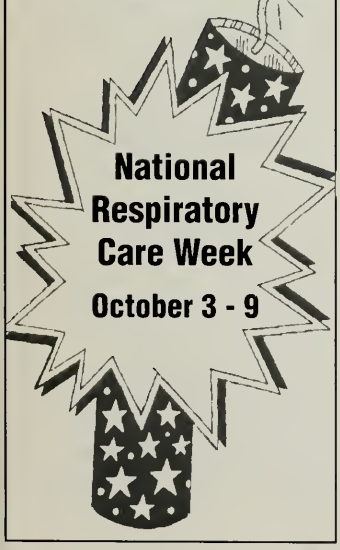

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- ***Diversity.*** With over 140 client hospitals in more than 40 states, Primedica has a place for you.
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- ***Education.*** Primedica's Education Department strives to keep our therapists current on the latest medical and technical advancements. Tuition reimbursement allows you to advance your degree. CRTT and RRT review courses are available for credentialing, and on-site teleconference and video conference programs keep you up to date on CRCE requirements.
- ***Opportunity to be a part of a professional team.*** Primedica people are making a difference in people's lives across the nation. We work to provide the very best Respiratory Therapy/Cardiopulmonary care possible. By striving to be the best, we have become a team "Providing Excellence in Patient Care."
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Visit AARC Booth #249, 251 and 253 in Nashville.

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Texoma Medical Center, a 250 bed regional referral facility in North Texas, is seeking a Director for our Respiratory Care Services. Qualified candidate must have a Bachelor's degree in Respiratory Therapy, or a related science, or the equivalent of work experience. Must be a Registered Respiratary Therapist and have 5 years of management experience.

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Texoma Medical Center is ideally located just one hour from Dallas and five minutes from Lake Texoma. Enjoy relaxed community living within close proximity to the metro area.

To learn more, call or send resume today to: Joy Wingfield, Employment Coordinator,
Texoma Medical Center,
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Denison, TX 75020
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ext. 4051.

At TMC, you can enjoy the excitement and challenge of a regional specialty center in the relaxed atmosphere of a small town.

TEXOMA MEDICAL CENTER

For more information, please call TMC Employment Coordinator

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The American Respiratory Care Foundation Awards for 1993

1. Dr Allen DeVilbiss Literary Award for the best paper published from November 1992 through October 1993 that addresses new technology or a new application of current technology in respiratory care: $2,000 cash plus travel expenses to the AARC Annual Meeting to receive the award.
2. $2,000 for the best original paper (study, evaluation, or case report) *accepted* for publication from December 1992 through October 1993. This award is *not* limited to papers based on OPEN FORUM presentations.
3. Four awards of $1,000 each for papers *accepted* for publication from November 1992 through October 1993 based on *any* OPEN FORUM presentation (not limited to 1992 OPEN FORUM).
4. Five awards of $500 each for the best papers *submitted* (not necessarily published) by 1993 OPEN FORUM participants who have 'never published' in the Journal. The never-published first author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the 1993 Annual Meeting (*received* in the Editorial Office by November 1, 1993). Co-authors may have previously published in RESPIRATORY CARE.

Three awards of $333 each are to be awarded to the authors of the three best features from Test Your Radiologic Skill, Blood Gas Corner, Kittredge's Corner, and PFT Corner accepted for publication from November 1992 through October 1993. All three (or none) of the features may be chosen from a specific category (eg, all three may be chosen from Blood Gas Corner).

All awards will be made at the 1993 Annual Meeting. Papers are judged automatically. No application is necessary.

Annual Meeting Registration Reimbursement

As in the past, any 1993 OPEN FORUM presenter (or co-author designee) will receive complimentary registration for an adequately prepared paper based on his 1993 OPEN FORUM abstract, submitted prior to or at the 1993 Annual Meeting.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1993 Examination and Fee Schedule

RPFT Examination

EXAMINATION DATE:	DECEMBER 4, 1993
Applications Accepted Beginning:	July 1, 1993
Application Deadline:	September 1, 1993

Perinatal/Pediatric Specialty Examination

EXAMINATION DATE:	MARCH 12, 1994
Applications Accepted Beginning:	July 1, 1993
Application Deadline:	November 1, 1993

Fee Schedule

Entry Level CRTT—new applicant:	$ 90.00
Entry Level CRTT—reapplicant:	$ 60.00
RRT Written and Clinical Simulation—new applicant:	$190.00
RRT Written and Clinical Simulation—reapplication:	$160.00
Written Registry Only—new applicant:	$ 90.00
Written Registry Only—reapplicant:	$ 60.00
Clinical Simulation Only—new & reapplicant:	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00
Perinatal/Pediatric—new applicant:	$150.00
Perinatal/Pediatric—reapplicant:	$130.00

	Active	Inactive
CRTT Recredentialing:	$25.00	$ 60.00
RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P Spec Credentialing:	$25.00	$130.00
Membership Renewal:		
CTT/RRT/CPFT/RPFT	$12.00	N/A

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Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

AARC & AFFILIATES

September 15-17 in Ocean, Maryland. The MD/DC Society presents its 12th Annual Conference by the Sea at the Carousel Hotel and Resort. For information, contact: Elgloria Harrison, PO Box 91753, Washington DC 20090, (202) 884-5105/884-5106, or Joseph Lynott at (202) 877-1064.

September 16-17 in Akron, Ohio. The Rehabilitation & Continuing Care and Cardiopulmonary Diagnostics Committees of the OSRC sponsor a Pulmonary Forum on Diagnostics and Rehabilitation. The seminar will be held at the Sheraton Suites. Featured speakers and topics include Bruce Sherman MD, "Handling Medical Emergencies"; James Stoller MD, "New Developments in Alpha-1 Antitrypsin Deficiency"; Colleen Servick RRT, "Designing Community Asthma Education Programs"; Cheryl Brown MHA, "Health Care Reform"; David Walsh RRT RPSGT, "Polysomnography"; Susan Rubaszewski RRT, "Maintenance Programs for COPD Patients"; Daniel Banks MD, "Challenge Testing for Specific Sensitizing Agents"; Ellen Hill MS RRT RPFT, "Developing Protocols for Pre-Admission Testing"; and Gene Butler JD RRT, and Kim Smith RN, "Caring for the Chronically Ill." Call (216) 498-1041 for additional information.

September 16-17 in Kingfield, Maine. The MSRC presents its Annual Fall Seminar at the Sugarloaf Resort. Topics include surfactant therapy, AARC consensus on education, pulmonary rehabilitation, effects of altitude on pulmonary system, and legislative updates on health care reform. Social events include a barbecue, dance, and golf tournament. Contact: Peter Leavitt RRT, St Mary's Regional Medical Center, Campus Ave, Lewiston ME 04240, or call (207) 777-8538.

September 22-24 in Duluth, Minnesota. The MSRC presents its 24th Annual Convention at the Duluth Entertainment and Convention Center. Topics include clinical practice guidelines and managing quality in health care. Approximately 45 vendors will display the latest technology. Social events include a golf tournament, volleyball, and an Octoberfest celebration. For more information, contact Kathy Prosnick at (218) 726-4543 or Steven Due at (612) 863-6890.

September 24 in River Grove, Illinois. The ISRC's Homecare Section and Triton College's Continuing Education Center for Health Professionals present the 5th Annual Homecare Conference, "Respiratory Homecare: Blueprint For the Future." Contact Shirley Kacena, Triton College, at (708) 456-8005.

November 12 in Nashville, Tennessee. The TSRC holds its Annual Meeting in conjunction with the Tennessee Hospital Association's convention and exhibition at the Opryland Hotel. Speakers/topics include Senator Robert Rochelle, "TennCare—Oversight Committee Update"; John Faulkner, president and CEO, Jesse Holman Jones Hospital, "TennCare's Impact on Hospitals"; Manny Martins, Director of Medicaid, "TennCare's Impact on Home Health and DME"; Roger Richardson RRT, "Medicare DMERC Consolidation"; and Dr Robert Tallon, "New DMERC Medical Policies." For information regarding registration and/or exhibits, contact Carol McCabe at (615) 256-8240.

OTHER

September 30-October 1 in Fresno, California. The Respiratory Care Department and Central Valley Foundation Valley Medical Center hold the 4th Annual Conference, "Controversies in Respiratory Care." Speakers/topics include Edward Abraham MD, "Inverse Ratio Ventilation: Effects on the Cardiac System"; William Dominic MD, "Surfactant Therapy for Pulmonary Injuries"; Alan Horowitz Esq, "Ventilator Alarms and Malpractice"; Enok Lohne MD, "Resurgence of TB: Is It An Epidemic?"; John Luce MD, "HIV and the Respiratory Care Practitioner"; Neil MacIntyre MD, "Controversies in Mechanical Ventilation"; and Michael Mahlmeister RRT, "PEP Therapy and Autogenic Drainage." For information and a brochure, contact Karen Boswell or Pete Williams at (209) 453-5197.

September 30-October 3 in Ann Arbor and Dearborn Michigan. The Extracorporeal Life Support Organization (ELSO) presents its fifth annual meeting, ELSO V and Advanced Course. The Thursday afternoon program will focus on economics, regulatory, and legal issues including a mock trial involving an ECMO case. New technology such as nitric oxide, fluorocarbon liquid ventilation, ventilation techniques for children and adults, and new applications of extracorporeal life support will be featured on Friday morning. The rest of the meeting will offer scientific presentations, committee meetings and

reports, round table discussions and state-of-the-art summaries. For information and registration contact Extracorporeal Life Support Organization, 1327 Jones Drive Suite 101, Ann Arbor MI 48105-1899. (313) 998-6600.

October 13 in Atlanta, Georgia. St. Joseph's Hospital of Atlanta presents its 10th Annual Respiratory Care Seminar. Contact: Rena Cobb RRT RCP, Education Coordinator, Respiratory Care, St. Joseph's Hospital of Atlanta, 5665 Peachtree Dunwoody Rd, Atlanta, GA 30342, or call (404) 851-7176.

October 14-15 in Asheville, North Carolina. The 5th Annual Mountain Air Conference for Respiratory Care features Dr Larry Pepper, manager of Shuttle Medical Operations for NASA, discussing innovations in health care delivery. Other topics include multidrug-resistant TB, hyperbaric therapy, stabilization of burn patients, and a pediatric asthma update. For more information, contact Charolette Artis at (704) 257-4473.

October 29 in Reno, Nevada. The American Lung Association of Nevada presents its 12th Annual Respiratory Health Conference in the Mack Auditorium at Washoe Medical Center. Contact: Sarah Kentfield, American Lung Association of Nevada, PO Box 7056, Reno, NV 89510-7056, or call (702) 829-5864.

April 2-8, 1994 in Miami, Florida. The 1994 session of Miami Children's Hospital's Annual VACC Camp for ventilation-assisted children and their families will be held. Application due date is Jan. 15, 1994. This community-based, free camp program provides recreation and social support for children with trachs, vents, CPAP, etc, and their moms, dads, and siblings at least five years old. Technology-dependent children should have normal cognitive potential. For information, contact: Director Moises Simpser MD, or Program Coordinator Cathy Klein, Ventilation Assisted Children's Center, Division of Pulmonology, Miami Children's Hospital, 3200 SW 60th Court, Suite 203, Miami, FL 33155-4076, (305) 662-VACC.

AARC 39th Annual Convnetion and Exhibition • AARC 39th Annual Convnetion and Exhibition • AARC 39th Annual Convnetion and Exhibition

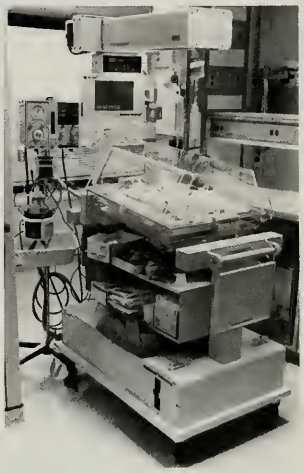
AARC 39th Annual Convnetion and Exhibition • AARC 39th Annual Convnetion and Exhibition • AARC 39th Annual Convnetion and Exhibition

New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229.

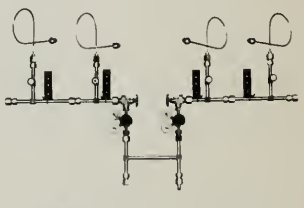
POCKET-SIZE MONITOR. According to the manufacturer, the peak-flow monitor, Spir-O-Flow features an exclusive dual measuring scale, allowing for both standard and low-range peak flow measurements. It is dishwasher safe, lightweight (2.7 oz), and meets the National Asthma Education Program technical standards. Spir-O-Flow is packaged 1 unit per box in cartons of 24 units (96 pieces) and comes with use and care instructions, mouthpieces and a daily record chart; a zip-lock pouch is available for storage. Contact Schuco Inc, Dept RC, 1419 Expressway Drive North, Toledo OH 43608. (419) 726-9000. Outside Ohio (800) 645-2500. Please mention RESPIRATORY CARE when you call.

ANTI-DISCONNECT DEVICE. Zipper Medical Products Inc introduces the first dual purpose ventilator anti-disconnect and trach-tie device (Model 201). This product (featuring a soft, cotton-lined trach tie with quick-release Velcro tabs) may be used wherever patients with tracheostomies are cared for. To receive a free sample, call (800) 647-0172. Please be sure to mention RESPIRATORY CARE when you call.



NEONATAL WORKSTATION. Vitaltrends Technology introduces the V_T1000 Neonatal WorkStation, offering noninvasive and continuous pulmonary function measurements without adding dead space or increasing airway resistance. The V_T1000 is a complete infant care unit incorporating the radiant warmer, patient bed, whole-body plethysmograph and conventional cardiopulmonary monitors. According to the manufacturer, a comprehensive, automated clinical information management system provides user-selected trend displays and reports from the more than 50 monitored and derived parameters tracked and stored. Vitaltrends Technology Inc, Dept RC, 145 Hudson Street, 8th Floor, New York NY 10013.

ISOLATION ENCLOSURE. Airo Clean Engineering is offering a new, 4-page, 2-color pamphlet that details its mobile isolation enclosure, also known as a bacteria-controlled nursing unit (BCNU). The BCNU, which can be set up in 30 minutes by 1 person, is described as a laminar airflow isolation cubicle with unidirectional air flow. The unit incorporates a physical barrier (transparent, flexible side walls), with two modes of operation: Containment and protection. For a free copy of the pamphlet, write Mike Pelosi III, Director of Marketing, Dept RC, Airo Clean Engineering, 212 Phillips Rd, Exton PA 19341.



GAS MANIFOLDS. Western Enterprises now offers INNOVATOR stainless steel manifolds designed for use in gas service systems requiring pur-

ities to 99.999%. The new manifolds feature 3/8", 316L stainless steel pipe and tee orbital-welded construction; 316L stainless steel primary regulators; an electropolished internal finish; check valves; pigtails; and a modular design that allows stations to be added without welding. Both HSAD and HSMS models can be equipped with alarms to signal near depletion of bank pressure. All models are intended for high purity gas applications requiring uninterrupted flows or greater on-line storage. Western Enterprises, INNOVATOR Sales Center, Dept RC, 875 Bassett Road, Westlake OH 44145-1196. (216) 871-2160. Mention RESPIRATORY CARE when you call.

EXTENDED-USE TANK. DependiCare Home Health Inc introduces extended-use portable oxygen tanks that enable respiratory patients to enjoy longer periods outside the home. According, to the manufacturer, the new oxygen tanks are smaller, lighter (4 lbs) and last longer (about 9 hours at 2 L/min flow) than other low-flow oxygen systems. They can be carried like a purse or back pack, with an over-the-shoulder strap. The gas supply depletes more slowly due to a pulse-dose, oxygen-conserving delivery system. Contact Sam Huff or Karen Yancey at DependiCare Home Health Inc (708) 382-7404. Please mention RESPIRATORY CARE when you call.

INSTRUMENT CART. The M4 cart from Midwest Products & Engineering Inc adapts well to many applications. The Tilt-Top M4 is designed to accomodate small to midsize medical or electronic devices and features 2.5" diameter casters, steel construction, and a convenient storage drawer. This 50 pound cart is shipped unassembled via UPS with assembly tools, hardware, and instructions included. Call Beth Culbertson, (414) 355-0310. Please mention RESPIRATORY CARE when you call.

SERVO INTERFACE. Innovative Biomedical Technologies Inc offers an accessory circuit board for the Servo 900C and 900D ventilators. This accessory captures 14 ventilator control and alarm settings and, when used with an analog-to-digital converter, collects dynamic patient data as well. The interface, designed for clinical information management systems. can improve the accuracy of charted data and decrease the time needed to record it. Contact Innovative Biomedical Technologies Inc at (612) 626-5586. Don't forget to mention RESPIRATORY CARE when you call.

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I. Type of Instn/Practice
1 ❑ Hosp ≥ 500 or more beds
2 ❑ Hosp 300 to 499 beds
3 ❑ Hosp 200 to 299 beds
4 ❑ Hosp 100 to 199 beds
5 ❑ Hosp <100 or less bed
6 ❑ Skilled Nursing Facility
7 ❑ Home Care Practice
8 ❑ School

II. Department
A ❑ Respiratory Therapy
B ❑ Cardiopulmonary
C ❑ Anesthesia Service
D ❑ Emergency Dept.

III. Specialty
1 ❑ Clinical Practice
2 ❑ Perinatal Pediatrics
3 ❑ Critical Care
4 ❑ Clinical Research
5 ❑ Pulmonary Function Lab
6 ❑ Home Care/Rehab
7 ❑ Education
8 ❑ Management

IV. Position
A ❑ Dept Head
B ❑ Chief Therapist
C ❑ Supervisor
D ❑ Staff Technician
E ❑ Staff Therapist
F ❑ Educator
G ❑ Medical Director
H ❑ Anesthesiologist
I ❑ Pulmonologist
J ❑ Other MD
K ❑ Nurse

V. Are you a member of the AARC?
1 ❑ Yes 2 ❑ No

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F ❑ Educator
G ❑ Medical Director
H ❑ Anesthesiologist
I ❑ Pulmonologist
J ❑ Other MD
K ❑ Nurse

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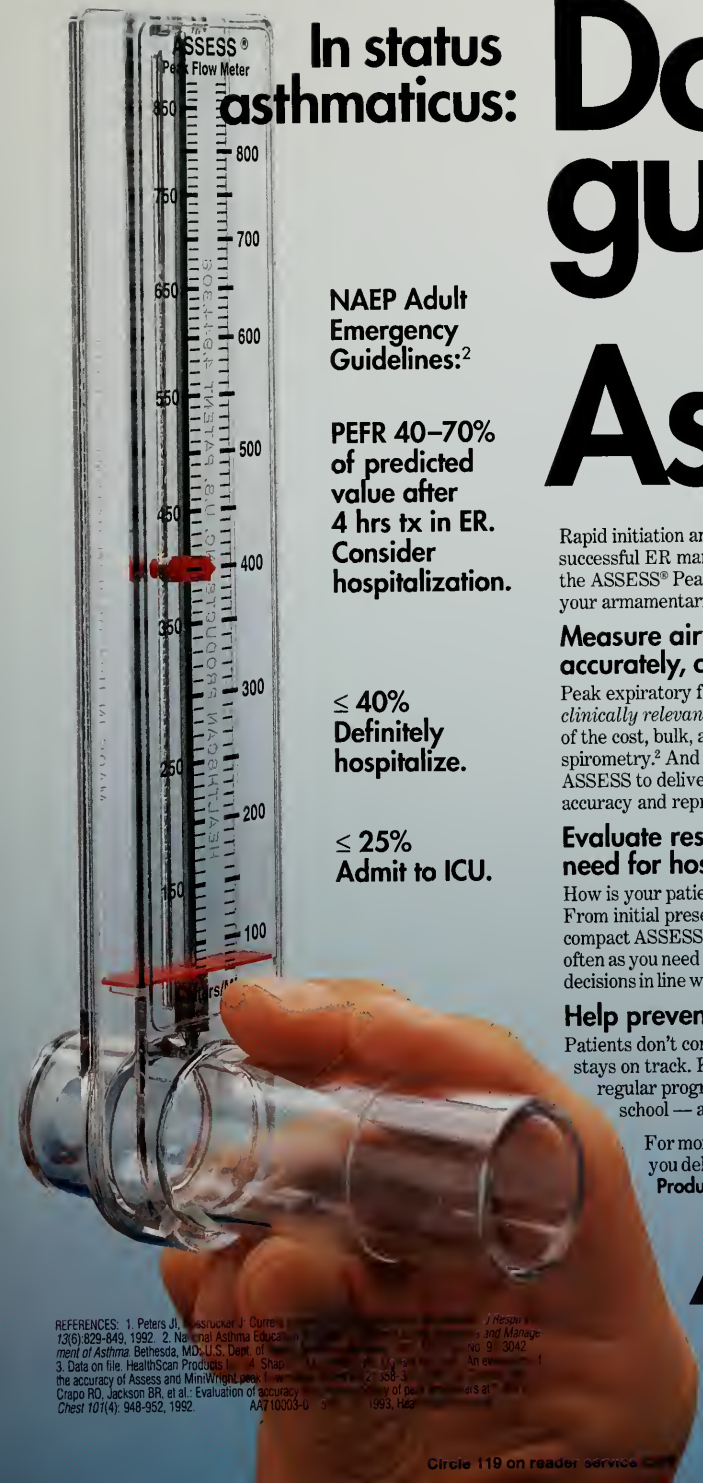

In status asthmaticus:

Don't guess.

Assess.

NAEP Adult Emergency Guidelines:[2]

PEFR 40–70% of predicted value after 4 hrs tx in ER. Consider hospitalization.

≤ 40% Definitely hospitalize.

≤ 25% Admit to ICU.

Rapid initiation and close monitoring of therapy are vital to successful ER management of severe asthma.[1] That's why the ASSESS® Peak Flow Meter should be a vital part of your armamentarium. ASSESS lets you —

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Peak expiratory flow rate (PEFR) provides an objective, *clinically relevant* measurement of airflow — at a fraction of the cost, bulk, and inconvenience of conventional spirometry.[2] And when seconds count, you can count on ASSESS to deliver those measurements with superior accuracy and reproducibility.[3-5]

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REFERENCES: 1. Peters JI, Strucker J. Curre... 13(6):829-849, 1992. 2. National Asthma Educa... ment of Asthma. Bethesda, MD: U.S. Dept. of... 3. Data on file. HealthScan Products I... 4. Shap... the accuracy of Assess and MiniWright peak f... Crapo RO, Jackson BR, et al.: Evaluation of accuracy... Chest 101(4): 948-952, 1992.

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